Historic, archived document

Do not assume content reflects current
scientific knowledge, policies, or practices.

a SF601
A145

May 1973

the origin and spread of
VENEZUELAN EQUINE ENCEPHALOMYELITIS

PROCUREMENT SECTION
CURRENT SERIAL RECORDS
FEB 17 '74
U.S. DEPT. OF AGRICULTURE
NAT'L AGRIC. LIBRARY

Animal and Plant Health Inspection Service · U.S. DEPARTMENT OF AGRICULTURE

415249

Prepared by

Veterinary Services

Animal and Plant Health Inspection Service

U.S. Department of Agriculture

Hyattsville, Md. 20782

CONTENTS

Page

Endemics and epidemics
- South America 1
  - Colombia and Venezuela 1
  - Ecuador 3
- Central America 3
- Mexico 4
- United States 7
  - Development of policy 8

Phase I—Pre-emergency plan 8
- The first case 9
- Holding operation 13

Phase II—Emergency plan 14
- Vaccination 14
- Vector control 15
  - Operations 17
  - Intelligence 17
  - Maintenance and logistics 17
  - Communications 18
  - Manpower-personnel 18
- Vector surveillance 18
- Environment 19
  - The region 19
  - Geography 19
  - Weather and climate 20
- Maintenance and spread 21
- Vaccination 22
- Viruses 23
- Vectors 25
  - Mosquitoes 25
  - Biting flies 27
- Morbidity and mortality 29
  - Hidden infection 30
  - Reporting 30
  - Clinical signs 31
  - Recovery after treatment 37
- Movement of horses, humans, and vehicles 37
- Disposal of dead horses 39
- Wildlife 39
  - Birds 39
  - Mammals 41

Discussion and conclusions 45

References 46

Appendix 48
- Historical chronology 48

THE ORIGIN AND SPREAD OF VENEZUELAN EQUINE ENCEPHALOMYELITIS

ENDEMICS AND EPIDEMICS

South America

The tangled vegetation and ink-black waters of the steamy, tropical rain forests of the Amazon and Orinoco may, it is theorized, be the seedbeds from which most of the arboviruses of the Americas arise. Fed by tributaries 1,500 to 2,100 miles long, the Amazon flows through the densest tropical forest in the world, much of it still unexplored, 3,900 miles from its source at Iquitos, Peru, to the Atlantic Ocean. Throughout its length the Amazon has a fall of less than 2.5 inches per mile. Since the margins of the continent are generally high and the interior low, drainage is away from the periphery toward the low levels of the Central river systems. This river network drains one-third of the South American Continent, an area equal to two-thirds that of the United States. The Orinoco, connected to the Amazon system, courses 1,700 miles north and east through Venezuela to the Atlantic Ocean. South America has more running water than any other continent.

In this humid, tropical rain forest, the median temperature in the coolest month exceeds 64°F., and in the driest month the rainfall is not less than 2.5 inches. The distinction between seasons is not wet and dry, but wet and very wet. Within this region favorable ecological conditions always exist somewhere for the maintenance of arthropod and vertebrate hosts capable of sustaining cycles of arbovirus infection.

As local conditions are modified by the immunization of susceptible populations or the relative abundance of insect vectors, infection shifts from place to place. Prevailing winds carry infected mosquitoes and biting flies rapidly over large areas. Infected migratory birds may further help disperse viruses great distances. In this manner, conceivably, the virus that was to produce encephalitis in equines and in humans was dispersed through South America.

Colombia and Venezuela

In 1935, an encephalitic disease of equines appeared in the river valleys of the departments of Huila, Tolima, Valle, and Bolivar in the Andes Mountains of western Colombia. The disease was tentatively diagnosed as Borna's disease, otherwise known as European Equine Encephalitis, an encephalitic, neurotropic virus disease primarily of horses and sheep, largely limited to certain areas in East and West Germany. It is suppositious but reasonable that this spill-out to equines and possibly to humans of vectors and virus from the swamps had occurred unheralded on previous occasions and in other localities. Nevertheless, the following year, 1936, the disease first appeared among equines in the department of Magdalena in northern Colombia. Later the disease emerged in La Guajira, the peninsula at the extreme northern end of Colombia that juts into the Caribbean Sea.

Shortly afterward the disease was recognized for the first time on the Venezuelan side of La Guajira, northwest of Lake Maracaibo. This coastal area is semidesert inhabited by a large population of burros. After infrequent rains massive numbers of *Aedes* mosquitoes appear. Two large swamps inland from the dry coastal area harbor cotton rats, raccoons, and opossums.

It is presumed that variations occurred in environmental conditions, in the small vertebrate populations, or in the relative abundance of vectors. These changes precipitated an overflow of virus from its maintenance cycle in the swamps into the burros in the arid area. Other agencies then helped spread the infection to susceptible hosts in new areas. In the next 3 years the virulent disease rolled across Venezuela eastward to the coast.

In 1938, Kubes and Rios *(23)* at the Laboratories of Veterinary Bacteriology and Parasitology, Ministry of

Agriculture and Animal Husbandry of Venezuela, Caracas, isolated a filterable virus from the brain of a horse that had died with encephalitic signs. The virus was immunologically different from, and more virulent than, the viruses of Eastern or Western Encephalomyelitis previously seen in South America. It caused death to guinea pigs within 48-72 hours regardless of the route of inoculation. Later in 1938, from portions of the brains of equines dying from the disease, Beck and Wyckoff, *(1)* at the Lederle Laboratories in New York, confirmed and amplified the characterization of the virus. Due to its origin, this equine neurotropic virus was designated "Venezuelan." (The next 30 years were to see the accumulation of a mass of confusing evidence of VEE viruses capable of producing different responses in various host animals in a dozen countries of North and South America. It was not until 1969 that some of the complexity was resolved with the classification of the VEE viruses into endemic and epidemic strains consisting of four principal types and five subtypes *(42*[1]*.)*

In 1943, an epidemic among equines, again originating in western Venezuela, moved eastward across the hot, dry lowlands to the coast.

From January to October, half a million horses were vaccinated with a chick-embryo vaccine developed in 1939 at the Institute of Veterinary Research in Caracas, Venezuela. On October 1, the disease appeared in 12 donkeys on the southern shore of the island of Trinidad, approximately 8 miles across the Gulf of Paria from Venezuela. A ring of equines immunized with the Venezuelan vaccine was quickly created around the disease focus to absorb the attacks of infected vectors. By March 1944, the vaccination in Trinidad of 6,000 equines around the infected area had apparently brought the epidemic under control.

VEE virus was isolated from *Mansonia titillans* mosquitoes infected in nature in Trinidad. It was theorized that *Aedes taeniorrhynchus* was the long-range carrier responsible for conveying the virus from the Venezuelan mainland to infect the first equine on Trinidad. Thereafter, *Mansonia titillans* spread the disease among equines on the island *(16)*.

The Guajira peninsula once again in 1961 and 1962, was the site for an outbreak of encephalitis and high mortality. Beginning in November 1962, a high incidence in children was reported. With its main thrust eastward across northern Venezuela, the epidemic lasted until May 1964. Almost 32,000 human cases with 190 fatalities were reported. VEE virus was isolated in samples of human sera and brain collected from the extreme northwestern to the northeastern corners of Venezuela from October 1962 to August 1964 *(2)*.

During the course of the epidemic, the Virology Department of the Venezuelan Institute of Scientific Investigations also sampled animals and insects in a number of localities where VEE in humans was occurring *(33)*. Mosquitoes were collected in November 1962 from the same locality in which the epidemic in humans began. VEE virus was isolated from lots of five species: *Aedes taeniorrhynchus*, *Aedes serratus*, *Anopheles aquasalis*, *Psorophora confinnis*, and *Aedes scapularis*. *Aedes taeniorrhynchus* was the most frequent species and the most frequent carrier of virus. Virus was not isolated from *Culicoides* spp. collected in two localities on two occasions. With regard to the second *Culicoides* collection, however, it was noted that mosquitoes collected at the same time and place likewise failed to yield virus.

From sera, hemagglutination-inhibition antibodies to VEE virus were detected in goats, sheep, opossums, cattle, pigs, and rats. Neither birds nor reptiles had significant antibody titres.

Analysis of climatic data revealed that during the 7 months preceding and the 4 months following onset of the epidemic the amount of rainfall in the area of the initial outbreak was double the average of the preceding 5 years. This provided ideal breeding conditions for mosquitoes and caused unusual crowding of animals on the remaining unflooded land.

Colombia is the only country in South America with a coastline along both the Atlantic and Pacific Oceans. Almost half of the country lies in the Andes Mountains, the remainder in tropical plains. Three cordilleras of the Andes running parallel north and south separate the Pacific Coast from inland valleys. Only in western Colombia (and in northern Ecuador) are both flanks of the Andes Mountains humid all the time.

Late in 1967, an epidemic of equines and humans appeared in extensive areas from the lower Cauca and Magdalena river valleys 950 miles north to the Atlantic coastal plain.

The first cases were recognized in a community near Cali where clay bricks were made. Removal of the clay from borrow pits had created numerous ponds. These were filled with vegetation and supported large numbers of mosquitoes.

Within 6 weeks, 38 percent of the equine population of 425 became clinically ill and 22 percent died. Subsequently 270 humans were stricken. VEE virus was isolated from 49 percent of the ill. There were no human deaths.

VEE virus was also isolated from animals: opossums, roof rats, Norway rats, a frugivorous bat, and a bird; as well as from mosquitoes—*Mansonia* (which was in greatest numbers and provided the most isolations), *Culex* (including *Melanoconion*), *Aedes*, and *Anopheles* (Vector Control Briefs, May 1969).

---

[1] Numbers in italics refer to References, pp. 46 through 48.

The epidemic near Cali ended abruptly.

The second community studied was approximately 38 miles from the first, situated in a semiarid valley. The principal industry, the manufacture of sugar cakes from cane, used a large equine population for transport. In comparison with the first community, mosquitoes were rare. Only two day-biting species were found on the horses: *Aedes sexilineatus* and *Wyeomyia*. (*Wyeomyia* breeds in the rainwater that collects in the leaf bases of Bromeliads, the tropical spiny plants to which the pineapple belongs). On the other hand, large numbers of black flies *(Simuliidae)* fed on both horses and humans during the day. Approximately one-third of the horse population died. VEE virus was isolated from both species of diurnal biting mosquitoes and from five species of black flies *(39)*.

In other locations, on open hillsides at elevations of 4,600 to 4,900 feet where equines were more widely scattered, epidemics appeared to be less acute but more prolonged.

It was estimated that between 50,000 and 100,000 equines died in Colombia during 1967 and 1968, and between 250,000 and 500,000 humans were affected *(26)*. In this area, not known previously to be infected, VEE virus was isolated from a sheep *(24)*. An outbreak of VEE affecting man, equines and young sheep appeared in the eastern part of La Guajira and the western part of Lake Maracaibo from October to December 1969.

Epidemic VEE reappeared in 1970 in the areas affected in 1967-68, as well as in localities previously unaffected. Partial responsibility for the resurgence of the disease was ascribed to the immunization of equines with the inactivated vaccines produced in Colombia and Venezuela. Moreover, it was believed that the epidemic of 1967, while varying in type and intensity, never really ended. It had merely changed character and had moved into other vertebrate and invertebrate hosts in nature. The inadequacy of the inactivated vaccines had been substantiated in 1967 and 1968 by spot checks in a number of different areas on several hundred equines previously immunized with inactivated vaccines. These revealed an absence of HI antibodies and deaths from natural infection of vaccinated animals in whom virus could not be detected. The inactivated vaccines, it was believed, were periodically inducing active disease *(26)*. Subsequently in 1968, 7,000 doses of a live attenuated VEE vaccine, known as TC-83, were acquired from the U.S. Department of Defense. This was an experimental vaccine developed by the U.S. Army Medical Research Institute of Infectious Diseases for the protection of humans in situations of high-risk exposure to the disease. The vaccine was the 83rd passage in tissue culture of the expidemic virus isolated from a clinically ill donkey in Trinidad in 1943. Its use in Colombia on more than 6,500 equines produced solid immunity without clinical effects. During the 1970 outbreak, 100,000 additional doses of TC-83 vaccine were acquired and administered.

Ecuador

Encephalomyelitis entered Ecuador, it is believed, from Colombia to the north sometime in 1941. Within the next 3 years the unidentified disease spread southward until it had become endemic along the entire Pacific coast. In early January 1945, a virus isolated from a sick horse 12 hours before death in the province of El Oro proved to be identical to the Venezuelan strain *(35)*. During this period the disease in Ecuador was not associated with high morbidity or mortality. Frequent instances were reported of improvement in equines following palliative treatment such as purges, bloodletting, or injections of urotropine.

Beginning in January 1969, an epidemic in equines and humans occurred along the coastal plain. Some equines were immunized with an inactivated guinea pig tissue vaccine produced in Ecuador. Others were vaccinated with the chick embryo product elaborated in Venezuela. The U.S. Air Force sprayed 150,000 acres with ultralow volume malathion at the rate of 3 ounces per acre. Larviciding with oil was also done.

In contrast with the 1941-1946 period, when equine morbidity and mortality were low, the epidemic of 1969 was believed responsible for the death of between 20,000 and 30,000 equines, a mortality rate of 90 percent.

There were no further reports of extension of the epidemic in South America. In late May or June 1969, however, equine deaths were reported in Central America.

Central America

From its detection in 1935, epidemic VEE had been incriminated in the death of hundreds of thousands of equines in South America. In Central America, on the other hand, there was neither record nor recollection of the occurrence of a VEE epidemic at any time in history. Sporadic equine deaths had been attributed to Eastern Equine Encephalomyelitis or to nonviral causes. In June 1969, this benign situation abruptly changed.

Deaths among equines were reported first along the Pacific coast of Guatemala near El Salvador. Shortly afterward deaths were noted from another focus in northeastern El Salvador. The disease then radiated from both foci. The principal immediate thrust was southeast through El Salvador and the Pacific Coastal plain of Honduras into northwestern Nicaragua.

The initial equine deaths in Nicaragua occurred during the first week in October. This placed the infection approximately 225 miles from the origin of the disease in Guatemala 3 months before.

Quarantines and restrictions against road travel were imposed, and a widespread equine vaccination program was undertaken to build immune barriers around areas of infection. The U.S. Army Medical Department provided more than half a million doses of TC-83 vaccine for use in Guatemala, Honduras, El Salvador, and Nicaragua.

By the end of October an estimated 3,000-6,000 equines had died in Guatemala, 5,000 in El Salvador, 500-700 in Honduras, and 300 in Nicaragua.

Except for some isolated cases in unvaccinated equines, the disease did not extend further south than the single northwestern province in Nicaragua in which it had appeared in October. In its northward extension along the Guatemalan coast the disease had been last reported in Retalhuleu, approximately 38 miles from the Mexican border. Vaccination, it was believed, had stopped the epidemic.

There was no ready explanation for the appearance of VEE in Central America. It was significant that the virus isolated during the epidemic was the same virulent I-B subtype found in Ecuador, and that the epidemic in Ecuador was still active at the time the disease appeared in Central America. There were two additional pieces of evidence for evaluation: individual surveys conducted from 1965 to 1968 by the Middle America Research Unit (MARU) and the Cornell University Medical College, and a joint survey by both institutions conducted during the epidemic of 1969 *(32)*.

First, the Middle America Research Unit, in 1965, had detected antibodies to VEE virus in humans living in the Atlantic lowlands of Guatemala and Honduras. The following year MARU found VEE antibodies in horses and in humans on the Pacific lowland.

Investigators from the Cornell University Medical College confirmed and amplified these findings in studies conducted from 1965 to 1968. Initially, virus was isolated from sentinel hamsters after exposure to wild mosquitoes. Subsequently, antibodies to VEE virus were demonstrated in humans, horses, and cotton rats located in the areas of Guatemala, Honduras, and British Honduras where VEE virus had been isolated from the sentinel hamsters.

The Cornell investigators enlarged the range of viral activity by isolating VEE virus from sentinel hamsters in the Atlantic lowlands of Nicaragua and the Pacific lowlands of Costa Rica.

Second, while the epidemic of 1969 was in progress, MARU and the U.S. Public Health Service conducted a joint survey on horses. Blood samples were collected from 62 horses in three locations 15-30 miles outside the epidemic zones and in which there had been no history of recent equine deaths. Nearly every horse had VEE neutralizing antibodies. This further substantiated the belief derived from the surveys of 1965-1968 that VEE virus was present and unrecognized in Central America before 1965 *(14)*.

The MARU and the Cornell studies together gave credence to the presence of VEE virus in Central America prior to 1965 and to suggest that the range of virus activity extended from Panama to Mexico.

The following August 1970, VEE appeared in Costa Rica on the Pacific coast near the southwestern border of Nicaragua. Equine deaths reportedly ended 8 to 10 days after vaccination. Deaths, it was noted, were unrelated to sex or age of the animals. It was also observed that human illness ceased shortly after equine infection was controlled by vaccination *(11)*. *Deinocerites pseudes* mosquitoes were implicated as vectors.

Mexico

The report in August 1969 of epidemic VEE in the upper Grijalva River Valley of Mexico near Guatemala was not the first evidence of VEE infection in that country. In 1962, neutralizing antibodies to VEE virus had been detected in a febrile 16-year old boy living in Champotón on the gulf coast of the State of Campeche *(29)*. Campeche abuts Guatemala on the north.

Ecological studies that had been conducted under the auspices of the Cornell University Medical College during July and August of 1963 and 1964, and at varying periods thereafter until 1967, provided considerable information about endemic VEE in southeastern Mexico.

Using adult hamsters as sentinels in mosquito traps, VEE virus was found in focal areas of lagoon, mangrove, and rain-forest habitats at Sontecomapan near the gulf coast. VEE virus was also found widely distributed through brushy and woody areas extending from Minatitlan and Coatzacoalcos to the border of Tabasco.

At Sontecomapan, virus activity was apparent in both dry and rainy seasons. Virus was less prevalent during the dry months in the brushy country from Minatitlan to Tabasco. The prevalence of virus and antibodies also diminished in the arid, irrigated region on the Pacific side of the Isthmus of Tehuantepec.

At Sontecomapan and environs, VEE virus was isolated from four genera of *Culex* mosquitoes, *Deinocerites pseudes*, *Haemogogus mesodentatus*, and *Wyeomyia mitchelli*. VEE virus was also isolated from opossums, bats, and the pooled tissues of 1 percent of resident birds. Antibodies to VEE virus were found in opossums, bats, cotton mice, black rats, a rabbit, a racoon, and a weasel. At Minatitlan, VEE virus was isolated from *Aedes scapularis* mosquitoes, and antibodies from cotton rats and about 3 percent of nestling juvenile herons.

Hemagglutination-inhibition antibody titres suggestive of natural infections were found in cattle and pigs (but not chickens) on the gulf coast. Again, as with other studies, the prevalence of antibodies to VEE was low in those same species in the arid, irrigated region on the Pacific side.

Clinical VEE in equines was recognized in northern Veracruz and southern Tamaulipas in June 1966. The outbreak coincided with heavy rains from June to September and an accompanying abundance of mosquitoes, wild rodents, pigeons, and aquatic fowl. Of approximately 1,000 sick horses, 30 percent died *(30)*. Retrospective studies conducted between September and November demonstrated positive hemagglutination inhibition reactions to VEE virus in 62 of 248 horses, 8 of 93 cattle, 1 of 4 fowl, and 7 of 14 cotton rats *(10)*.

During 1967 and 1968, isolated cases of clinical VEE in horses were reported near the cities of Tampico and Tuxpan, Veracruz. A total of 18 deaths occurred.

Beginning in May 1969, unconfirmed reports were received of equine illness and death near the Mexican-Guatemalan border. In August, the first official reports appeared concerning deaths following signs suggestive of VEE on a ranch near Socoltenango in the upper Grijalva River basin of the State of Chiapas.

Chiapas, the southernmost state in Mexico, borders and lies mostly west of Guatemala. The Grijalva River forms a valley 20-35 miles wide running northwest from Guatemala through Chiapas. The river valley is a topographical extension of valleys in Guatemala where severe outbreaks of VEE had occurred in 1969.

From single, unpaired serum samples, scientists at the Instituto Nacional de Virologia, Mexico, D.F. detected antibodies specific for VEE. Field investigations revealed additional cases near Chicomucelo, Frontera Comalapa, and La Concordia, all in the upper Grijalva River Valley. This was the first recorded VEE epidemic in the Grijalva River Valley. Seemingly an extension of the epidemic from adjacent Guatemala, the disease persisted until November. Twenty-seven ranches were involved with a total of 150 equine deaths.

Then, beginning in November, isolated cases of clinical VEE were reported in four additional contiguous municipalities in the Grijalva River Valley. By the end of February 1970, approximately 300 equines had died. The majority of cases were on premises along the Grijalva River. *Aedes*, *Culex*, and *Anopheles* mosquitoes and *Simulium* (black flies) were reported present. VEE virus was isolated and identified from samples of human and of equine sera at the Instituto Nacional de Virologia.

There were no further reports of equine deaths due to VEE from March until May. With the start of the rainy season in June the disease reappeared in the Grijalva River Valley. Sera tested at the Instituto Nacional de Virologia revealed antibodies to VEE in 300 clinically-well equines. Shortly thereafter epidemic VEE virus was isolated from equines in the same area of the Grijalva River Valley. Following the laboratory confirmation of epidemic VEE, an immunization program with TC-83 vaccine was begun.

In the meantime, the disease had spread westward through mountain canyons to Salina Cruz and Tehuantepec on the Pacific coast. Malathion at ultralow volume was spot-sprayed in Chiapas and Oaxaca during August. The project was discontinued at the end of the month due to increasing costs as the area of infection enlarged.

An attempt was made to halt the spread of the disease eastward by vaccinating equines from Minatitlan, Veracruz, to Salina Cruz, Oaxaca, on the Pacific coast. Within 7 weeks, 418,000 equines had been vaccinated in the States of Chiapas, Oaxaca, and Veracruz. Equine mortality in the problem areas allegedly ended 7 days after vaccination. This did not, however, stop the spread of VEE into unvaccinated areas.

In the middle of September the disease appeared among horses of an army regiment stationed at San Andres Tuxtla, Veracruz, outside the vaccination zone. The vaccination belt was extended, only to be followed by the appearance of the disease north of the new vaccination barrier. This procedure was repeated several times until November when temperatures dropped and the heavy rains ended. Vaccination at that time had reached Nautla, Veracruz.

On the Pacific coast, the disease had followed a similar course, continuing to leap beyond each extension of the vaccination belts. By mid-November, VEE had spread from the Isthmus of Tehuantepec north along the coasts of the States of Oaxaca and Guerrero to the mouth of the Balsas River southeast of the State of

Michoacan. Vaccination was extended up to the State of Colima.

Approximately 10,000 equine deaths attributable to VEE were recorded in the States of Chiapas, Oaxaca, Veracruz, Guerrero, and Michoacan during the year 1970. This figure, however, was believed to be an underestimation. Owners reported losses at the vaccination centers. But frequently, individuals who owned only one or two equines did not go to the center to report their losses.

Early in April 1971, it was evident that VEE was again on the move in Mexico. Equine illnesses and deaths were reported in the vicinity of Lake Tamiahua in northern Veracruz, the same area in which outbreaks of VEE had occurred in 1966. Within a matter of days, 140 equine deaths were reported. The U.S. Department of Agriculture (USDA) requested the concurrence of Mexico to send a specialist to work with the local authorities so that a firm laboratory diagnosis could be made by virus isolation. Mexico concurred and a confirmed diagnosis was subsequently made. Field investigations were conducted on affected ranches located between Papantla and Tampico Alto. These revealed that sickness and death had not subsided in November, as believed, but in fact had continued unreported throughout the winter.

On a large ranch in Papantla, for example, unreported sickness and death in a herd of 136 equines had occurred from October through April: in December (five sick, three dead); February (two sick, two dead); March (six sick, five dead); April (two sick, two dead).

On "La Charolesa" ranch north of the mouth of the Tuxpan River, among 38 equines, three had sickened and one had died. Blood and nasal exudate from one of the sick horses was injected into each of two hamsters in the field. Both hamsters died. The brain of the hamster injected with blood was harvested and in various dilutions injected into additional hamsters. These died with the sample diluted up to 10^{-7}. Subsequent neutralization tests and virus isolations from the brain of one of the hamsters confirmed the disease as epidemic VEE. Epidemiologically significant was the fact that the three sick horses were part of a shipment of six that had been imported in January from northern Tamaulipas.

On April 14, at a ranch near Tampico el Alto, hamsters died after injection with blood from sick horses. Blood from a veterinarian (with a fever and flu-like symptoms) who had been working with sick horses was also injected into a hamster. The hamster died 5 days later. Epidemic subtype IB subsequently was isolated from the samples obtained from two of the horses and from the veterinarian.

In an effort to control this latest epidemic, 140,000 equines were immunized in an extension of the vaccination zone from Nautla, Veracruz, to Soto La Marina, Tamaulipas. The Ministry of Agriculture and Livestock requested protocols and TC-83 seed virus from the U.S. Department of the Army. Upon their receipt, the National Institute of Livestock Research started to manufacture the live attenuated VEE vaccine at Palo Alto. (The first 50,000 doses were ready for use in northern Tamaulipas on July 3).

From August 1970, when the massive vaccination program in the Grijalva River Valley began, until April 1971, 700,000 equines had been vaccinated with TC-83 vaccine. If vaccination had arrested mortality among equines in affected areas, as was attested, it had obviously failed to prevent the emergence of VEE beyond the vaccination areas. Admittedly, not all equines had been vaccinated. Allegedly, the more valuable horses were favored over other equines.

In the next several months, though reports were tentative and evidence inconclusive, what was believed to be VEE consolidated, gathered momentum, and like a tropical storm swept northward across the salt water lagoons of Mexico's gulf coast, spreading sickness and death in a swath 6-10 miles wide and 250 miles long.

The entire VEE problem was explored in Mexico in mid-May at a meeting of the Pan-American Health Organization. Opinion was divided on whether additional research was needed on the TC-83 vaccine. Some believed that vaccinated horses were the source of spread of virus beyond the periphery of vaccinated areas. Nonetheless, basic agreement was reached on a cooperative undertaking to protect the equine population of Mexico and the United States by enlarging the barrier of vaccinated animals. In anticipation of the early start of the program, the U.S. Department of Agriculture on May 23, shipped 200,000 doses of TC-83 vaccine to Mexico.

The agreement was consummated in June. By its terms Mexico agreed to provide and pay the salaries of personnel. The USDA agreed to supply sufficient vaccine to immunize the estimated 200,000 equines in 28 municipios of Tamaulipas and 42 municipios of Nuevo Leon. The Department of Agriculture also agreed to pay for gas and oil and for per diem expenses of the Mexican staff. Vaccination started on June 12.

The General Office of Animal Health assigned 15 teams to the vaccination program. Each consisted of a veterinarian and his assistant equipped with a vehicle. Immunizations started in the municipios closest to the Mexico-United States border and proceeded south. But expectations exceeded accomplishments and by mid-June clinical encephalitis and equine deaths were being reported i the San Fernando area and in coastal villages 35-50 miles south of H. Matamoros.

Then on June 25, sick horses showing incoordination and inappetence were reported in Ejido Palo Blanco, a commune of 181 persons and 40-50 houses in northern Tamaulipas. Although the area around Ejido Palo Blanco was cultivated, heavy rain had left abundant water in large and small pools. Mosquitoes had been so bothersome, particularly at night, that residents had been using smoke to discourage attacks.

On July 1, all horses, including those already sick, were vaccinated. Between 1 and 2 days later, several horses were showing clinical signs of VEE. These included braced stance, immobility, and temperature of 106.6°F. Recumbency and violent thrashing followed. Death usually came less than 24 hours later. Of a total horse population of 19, seven died.

Ejido Palo Alto was located 12 miles west of H. Matamoros and about 7 miles southwest of the United States border.

United States

Scientists in the United States had been following the progress of VEE since its discovery in South America and its corroboration as a new viral disease by Beck and Wyckoff in 1938 *(1)*. Despite this interest, as well as the finding in 1955 of VEE antibodies in three migrant Louisiana waterfowl, VEE was believed to be a disease limited to the tropical Americas. Then in April 1960, scientists documented evidence of the first human infection from a VEE virus in Florida. During a serological survey for arbovirus infections, VEE antibodies were detected in 7 percent of Seminole Indians living on the Brighton Reservation, and in 58 percent of those living on the Big Cypress Reservation 35-80 miles north of the Florida Everglades *(41)*. Viruses could not be isolated from several thousand mosquitoes collected at that time in the same area.

Three years later, VEE antibodies were found in 56 percent of Miccosukee Indians living along the northern border of the Everglades. VEE virus was isolated (in 1963 and again in 1964) from mosquitoes collected in a variety of habitats including swamps, pinelands, and hammocks in Everglades National Park. Three isolations of a virus apparently identical to that of VEE were made from *Culex* mosquitoes of the subgenus *Melanoconion* collected on a hardwood hammock. Cotton mice *(Peromyscus gossypinus)* and cotton rats *(Sigmodon hispidus)* trapped in 1964 on the same hammock on which the mosquitoes had been collected showed significant HI antibody titres against a Colombian strain of VEE virus *(5)*. Antibodies to VEE virus were also detected in the rice rat *(Oryzomys palustris)*, the black rat *(Rattus rattus)* and the white-tailed deer *(Odocoileus virginianus)*, as well as from cotton mice and cotton rats in agricultural areas and sawgrass marshes east of the Everglades National Park. This region was removed from the hammock environment with which VEE previously had been associated.

During the 1960's in Utah, complement-fixing antibodies to VEE were detected in eight species of wildlife, including mice, squirrels, rabbits, and fox, and seven of 4,719 range cattle. The desert environment of west central Utah was completely foreign to the usual habitat of endemic VEE in moist swamps.

Clinical VEE appeared in a human female from Homestead, Fla., in September 1968. In three successive bleedings the patient's sera showed rising antibody titres to the Florida strain of VEE virus. Virus was not detected in mosquitoes collected near the patient's residence. However, VEE virus was isolated from mosquitoes collected for 2 months following the reported infection, in other areas of the county where the patient frequently had fished. In a subsequent serological survey hemagglutination-inhibition antibodies to VEE were detected in six additional persons who lived or worked within 2 to 12 miles of the area *(12)*.

It was evident that the Florida strain of VEE virus was capable of causing both subclinical and clinical infection in humans. What was not so evident was whether VEE virus in southern Florida arose from a longstanding focus of infection deep in the Everglades, sporadic and expanding under favorable conditions; or whether it had been introduced by some unknown mechanism of dissemination from endemic-epidemic foci in South America, Central America, or Mexico.

Since VEE had never been reported in horses in Florida or any place else in the United States, two questions arose: Was the absence of horse involvement due to lack of contact between horse and virus, or did the Florida strain lack affinity for the horse? Experimental evidence indicated that the Florida strain of VEE was avirulent for horses *(18)*. The failure of the virus to develop in sufficient levels in the blood of horses in effect eliminated the horse as a disseminating or amplifying host, and limited the disease cycle to mosquitoes and small mammals.

The report of a suspected epidemic of VEE in Guatemala in June 1969 brought a prompt response from USDA. On July 7, two veterinarians from the Animal Health Division, Agricultural Research Service, arrived in Guatemala to establish a joint effort with other agencies of government. These included, in addition to the Animal Health Division, representatives from the U.S. Department of Defense, the U.S. Public Health Service, the Ministeries of Agriculture and the Ministeries of Health of both Guatemala and El Salvador, and the U.S. AID Missions to Guatemala and El Salvador. This experience led to a reappraisal of U.S. policy with respect to VEE.

Development of Policy

As a result of the cooperative experience in Guatemala, USDA started to develop a set of recommended procedures to be followed in the event of an outbreak of VEE in the United States. Meetings were held among Divisions of the Agricultural Research Service (ARS) and with representatives of the U.S. Army Medical Research Institute of Infectious Diseases, the U.S. Public Health Service, and the equine industry. The problem was also discussed in Mexico with key spokesmen for agriculture and health of several Central American countries, the Ministry of Agriculture and Livestock of Mexico, and consultants of the Pan-American Health Organization.

Four basic considerations influenced the development of ARS policy with respect to VEE. These were: (1) the existence and immediate availability of an organization of veterinarians and technicians familiar with combating animal diseases; (2) longstanding precedents concerning foreign diseases; (3) the peculiar epidemiology and natural history of VEE; and (4) the necessity of evaluating the possible consequences of any action upon both the public health and the best interests of the equine industry. Ultimately, the interchange of agency views produced a workable understanding.

Initially significant in the deliberations was the fact that at least one strain of virus was already in the United States. It was believed to be less virulent than the strains seen in Central and South America. Conceivably an epidemic of VEE in the United States could arise from conversion of the mild endemic strain or from introduction of a virulent strain from Latin America. Since clinical VEE had never been detected in equines in the United States, it was considered to be a foreign disease. The Agricultural Research Service had long maintained a policy of not licensing the production of biologics for livestock diseases foreign to the United States.

The only vaccine available was the modified live-virus vaccine, TC-83, developed by the Department of Defense. (Intended for the protection of laboratory workers, TC-83 was not licensed for general use in humans.) License to produce live-virus vaccines for use in animals is granted after demonstration of efficacy, safety, and nonreversion to virulence of experimental vaccines in controlled field trials. Despite commendable verbal reports about TC-83 during epidemics of VEE in Central and South America, its efficacy and safety had not been tested under controlled conditions. And while the possibility of reversion to virulence of the vaccine had been investigated in small laboratory animals, it had not been studied in horses. Under the circumstances, the vaccine was considered experimental.

Granting a license to manufacture TC-83 for use in the United States consequently depended upon demonstration of its safety and efficacy in controlled field trials with horses. Permission to use the vaccine further depended upon the concurrence by the U.S. Public Health Service that the vaccination of horses with live VEE virus would not endanger the human population. Finally, the use of TC-83 would be authorized only if epidemic VEE occurred in the United States.

Since TC-83 vaccine was already being used as well as manufactured in Mexico under U.S. Department of Defense protocols, USDA and the General Office of Animal Health of Mexico agreed that the most expeditious evaluations could be accomplished there. Testing for safety and efficacy, including backpassage in horses, would be done at the National Institute of Livestock Research, Palo Alto.

The final step in the formulation of policy was a request to the Department of Defense to reserve for USDA at least 1 million doses of TC-83 vaccine. This would be used in the event other control measures failed and an outbreak of VEE occurred among equines in the United States.

PHASE I—PRE-EMERGENCY PLAN

The rapid, unchecked progress of VEE through Mexico was causing apprehension in all quarters in the United States. Human health was in peril. In imminent jeopardy, however, were the estimated 625,000 horses in Texas, and looming not far behind, a population of 7 1/2 million horses representing an industry valued in excess of 12 billion dollars.

To combat this accelerating threat, a task force of multidisciplinary specialists began to assemble in Harlingen, Tex., on June 19. The task force objective was to protect horses and humans from VEE, and in the event of its appearance in the United States to implement the necessary control measures to halt the spread of the disease. The team included representatives from the

USDA, the U.S. Public Health Service, the Texas Animal Health Commission, and the Texas State Department of Health.

According to authorities, reports of the rapid spread of VEE in Mexico was producing a mass exodus of horses out of southern Texas. Many horse owners and veterinarians favored immediate release of the vaccine. This, it was believed, would stop the panic.

Within the next several days the task force made contingency plans for a massive vaccination and mosquito abatement program along the United States-Mexico border from Brownsville to Sullivan City, Tex.

In view of the questionable disease situation in Mexico and the unease this was creating in Texas, the USDA modified its policy and on June 23 released TC-83 vaccine.

On the afternoon of June 23, the USDA, in an agreement with the State of Texas, released vaccine TC-83 for voluntary use in 13 southern counties. Under the agreement, the Animal Health Division, ARS, was to supply the vaccine. Accredited veterinarians would administer the vaccine within the prescribed area of 13 counties south of a line drawn from Corpus Christi to Laredo. The target population contained an estimated 25,129 equines in 4,157 herds.

Although 2 million doses of TC-83 had been used to control epidemics among equines in Central and South America and in Mexico, the vaccine was still considered experimental and was unlicensed. Consequently, participating horse owners had to agree to waive claims for damages against the United States and its agents, not to move vaccinated equines for 14 days, and to report adverse reactions in vaccinated animals.

The voluntary vaccination program started on June 25. Vaccine was distributed free to veterinary practitioners at five distribution centers. Horse owners reimbursed practitioners for their time and services. Concurrently, an extensive public information program was disseminated via all the news media and the county extension service. (Information alerts to livestockmen actually had started in March.) These programs included information concerning the control of vectors on animals and on premises, how to get horses vaccinated, experiences with the vaccine in other countries, the limitations of the vaccine, the reactions horses might exhibit after vaccination, the manner of reporting sick animals, and the implications of the waiver of claims. Diagnosticians and support personnel were stationed at each distribution center to investigate all reported cases of sick equines.

Within a week of its formation the VEE Task Force at Harlingen had been transformed from an evaluation group to an action group engaged in five principal activities: (1) surveys of mosquito populations, (2) census and surveillance of equines, (3) diagnosis of VEE, (4) voluntary vaccination, and (5) field trials to evaluate the effectivity of the vaccine.

The First Case

In the sequence of events, the first sick horses in the lower Rio Grande Valley were reported to task force personnel on June 23, the same day the USDA officially released TC-83 vaccine. Several race horses among 10 in a boarding stable 5 miles north of Brownsville exhibited signs of depression, respiratory distress, and were coughing. Some had ocular discharges. A stallion had a temperature of 103°F. These cases did not cause undue alarm. They were considered part of a widespread "distemper-strangles-influenza" syndrome prevalent in southeastern Texas at the time.

Then, as if activated by the investigation at the stable near Brownsville, suspect equine illness began rapidly to appear at near and distant places.

That same day, June 23, on a horse breeding ranch near Three Rivers, Live Oak County, approximately 200 miles northwest of Brownsville, an unvaccinated 3-month old colt sickened. The colt failed to respond to treatment. Signs of central nervous system involvement appeared: a drifting gait, crosslegged walk, hanging head, and reaction to pressure between the eyes and poll. The animal's temperature was 105°F. The colt and its mare were taken to a veterinary hospital. The colt died during treatment on June 30. Samples of serum and brain tissue were carried for analysis to the Communicable Disease Center (CDC) at Atlanta, Ga.

Investigation revealed that the colt had been born on the ranch and until its terminal trip to the veterinary hospital had never been off the premises. Five mares had been brought to the ranch for breeding and had remained 2-3 months. One mare returned to its home premises in San Antonio (80 miles northwest of Three Rivers) 1 month before the colt became sick. At about the time the colt became sick, two mares were returned to their owners in San Diego (61 miles south of Three Rivers), while another mare was returned to its home in Falfurias (87 miles south of Three Rivers). The fifth mare was returned to its owner in San Antonio several days after the colt died.

Only the stallion had been vaccinated (in March 1971) for both Western and Eastern Equine Encephalomyelitis. None of the 26 horses on the ranch had been vaccinated against VEE. Examination of the herd at the time of the colt's death revealed "no signs of disease with central nervous system involvement." The ranch owner acknowledged having had "strangles" in his herd prior to June 24.

On July 1, a sick 3-year old mare was reported at Brownsville. A task force diagnostician seeing the mare for the first time that day observed a hanging head, signs of mastitis and septicemia, and a temperature of 105.4° F. All reflexes, it was noted, were normal and there were no signs of encephalitis or central nervous system involvement. Nevertheless, a blood sample was taken for testing at the CDC laboratory. (A second sample was obtained on July 6.)

On July 1, six of the horses on the premises, excluding the sick mare and her colt, were vaccinated against VEE. The mare had been vaccinated against both Western and Eastern Encephalomyelitis in May 1971.

The tests were completed and publicized on July 9. Epidemic (subtype IB) VEE virus had been isolated and identified from the sample taken on July 1. Hemagglutination-inhibition antibodies against VEE had been found in the sample taken on July 6. The titre was 1:1280 (as compared with 1:80 against EEE and 1:160 against WEE).

VEE virus in the first blood specimen and the high level of VEE antibodies in the later sample were evidence of VEE virus infection in the mare.

The isolation of epidemic VEE virus from the brain of the colt at Three Rivers was also confirmed on July 9.

Officially Venezuelan Equine Encephalomyelitis was now in the United States. It was apparent that VEE had slipped through the vaccination barrier as it had so often done before in Central America and in Mexico. The almost simultaneous appearance 200 miles apart of the first two recognized cases of VEE in the United States (fig. 1) was, in effect, a repetition of the origin and spread of the disease in the Western Hemisphere.

Since the use of TC-83 vaccine had already been authorized, confirmation of the first case as a condition precedent to its use was academic. Identification of the first case, however, was important epidemiologically. It not only presented an environmental prototype for early VEE in horses and in humans in the lower Rio Grande Valley, but demonstrated the difficulties in the precise determination of the extent and degree of the infection.

To take the second point first. When the mare was first seen by a diagnostician on July 1 there was nothing in the visible signs to arouse suspicion that this was anything but a case of mastitis and septicemia. On that basis, if this case had occurred in late summer the investigation probably would have ended with the first visit. Since the owner was persistent and it was one of the first cases, paired blood samples were taken. The implications were not apparent until a complete epidemiological investigation was made subsequent to laboratory confirmation of VEE.

The first signs of sickness were seen about June 15, 3 weeks after the mare had foaled. As reported by the owner, the mare acted "colicky," continually lying down, getting up, and lying down again. She refused to eat and was depressed. Later the mare stood stiffly immobile for several days and her pasterns swelled. The mare appeared to be in heat and repeatedly discharged small amounts of urine. A private veterinary practitioner treated the mare on June 20 and several times thereafter. It is possible that the signs suggestive of a central nervous system disturbance seen by the owner after June 15 (but not by the diagnostician on July 1) were the beginnings of VEE. (Some authorities believe viremia is long-lasting, persisting even until death.)

Although the colt at Live Oak was the first official investigation from which a sample confirmed as epidemic VEE was obtained (table 7), evidence indicates that epidemic VEE made its initial entry in the Brownsville area. Laboratory tests subsequently confirmed epidemic VEE virus in mosquitoes collected at San Pedro, Tex., on June 28 (table 6). In point of fact, as events were soon to demonstrate, many horses in different locations were apparently incubating VEE at about the same time in southern Texas.

The first signs of recovery in the mare were noted on July 8 when she began to eat and to drink water.

The D.P. premises on which VEE was diagnosed comprised 8 acres with one side bordering a horseshoe-shaped resaca about a mile long and 60 yards wide (fig. 2). The portion of the resaca adjoining the premises was clogged with vegetation in puddles of shallow, stagnant water. Depressions in the terrain, roadside ditches, and animal hoofprints in the caliche-clay soil had become pools of standing rainwater. A concrete irrigation ditch with several surface outlets for irrigation extended across the width of the property. Mosquitoes reportedly were so numerous from January to the middle of June that residents were unable to sit out of doors at night. Tabanids were present at all times. When there was ample water in the resaca several dozen ducks became residents. Twenty to 30 cattle egrets perched on the cattle and horses to eat insects.

Two horses were kept in a corral 15 yards from the resaca. The sick mare and her colt were in a separate corral adjacent to the owner's house also located 15 yards from the resaca. Four horses, together with one heifer, were pastured in a field of weeds and wild sunflowers. One end of the field where the animals customarily gathered to be fed was a large, deep mudhole. The horses were fed pellets of composite ingredients.

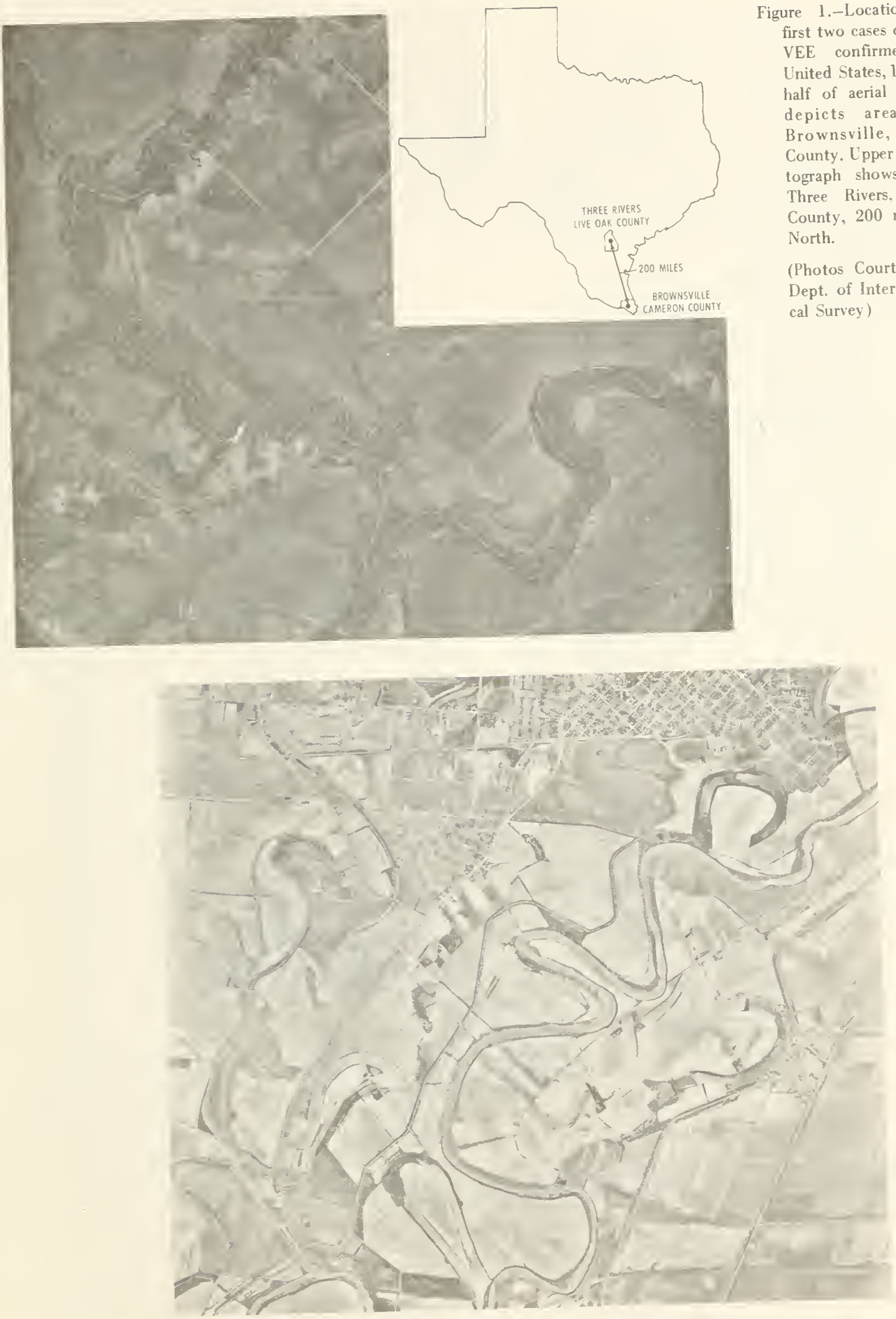

Figure 1.–Location of the first two cases of epidemic VEE confirmed in the United States, 1971. Lower half of aerial photograph depicts area west of Brownsville, Cameron County. Upper half of photograph shows region of Three Rivers, Live Oak County, 200 miles to the North.

(Photos Courtesy of U.S. Dept. of Interior, Geological Survey)

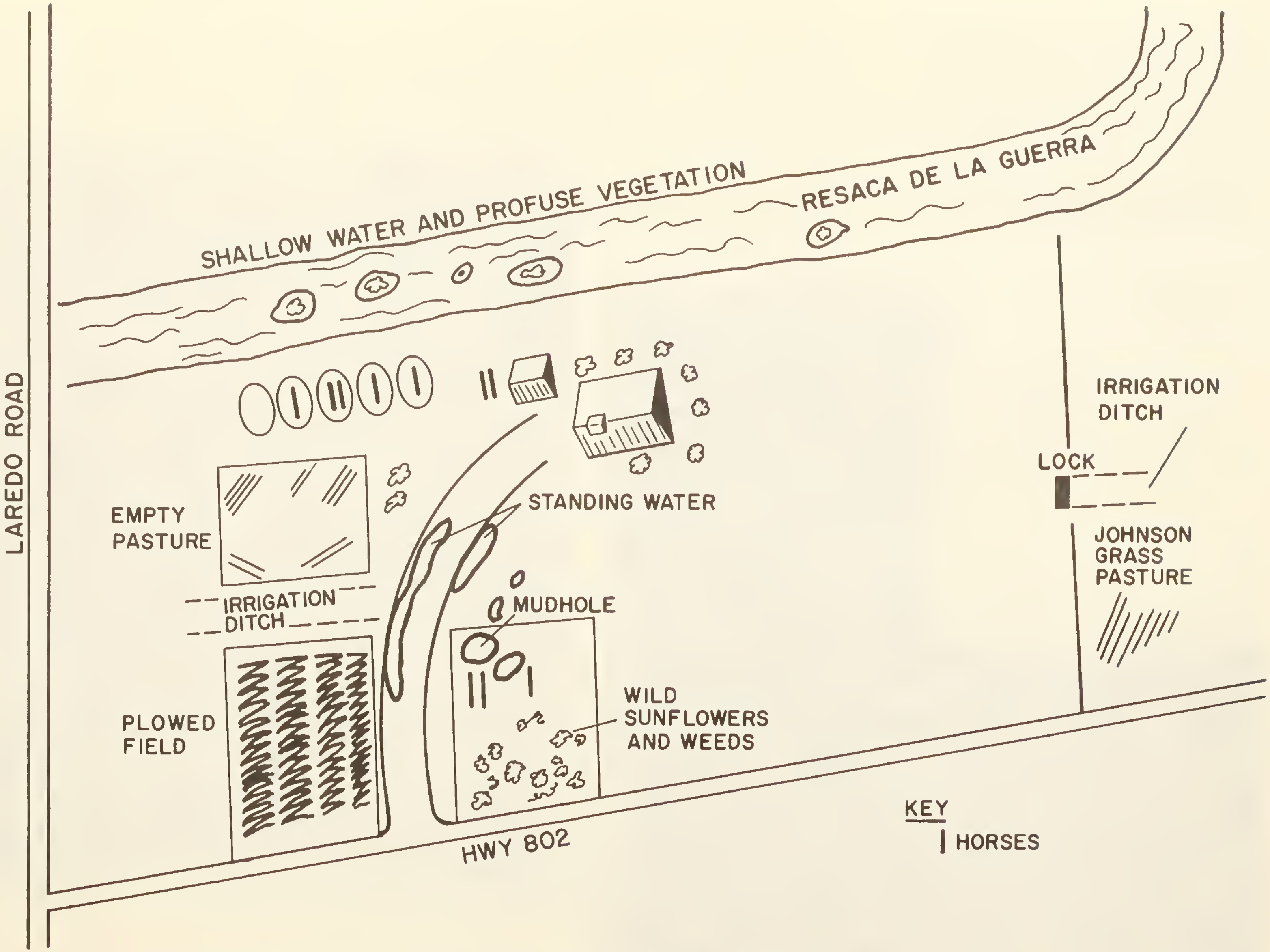

Figure 2.–Site of the first case of epidemic VEE confirmed in Brownsville, Cameron County, Tex., 1971.

Five of the eight horses belonged to owner D.P. The remainder had been boarding on the premises for 4 to 6 months. Periodically they were ridden on and off the property. Neighbors with horses had been on the premises several times in June.

In addition to the heifer, there were two dogs, five cats, and several dozen young chickens ages 2 days to 2 months of age on the property. On July 4, 2-month old chickens suddenly began to die. This was accompanied by deaths in the large blackbird population. The deaths among chickens ended in 5 days without a definitive diagnosis.

Another matter of epidemiological interest concerns a neighbor, J.G., an employee of an area water district who had been working part-time on the D.P. premises. His duties, at least once a week during the previous 6 months, included building fences, roping horses for treatment, and grooming. At about the time the mare became sick, J.G. complained of illness but continued to work.

J.G. owned two horses which he kept in a stall at his house half a mile away. One of these horses, an 8-year-old gelding, had been losing weight for 2 weeks. The horse was wormed but became progressively worse. Some of the signs noted were: depression, inappetence, braced stance, difficulty in breathing, a cough, drooling, ocular discharges, and impaired vision.

On July 4, the owner of the mare vaccinated J.G.'s sick gelding against Western Equine Encephalomyelitis and Eastern Equine Encephalomyelitis. The vaccine was injected subcutaneously instead of intradermally. A task force diagnostician was called on July 5. The gelding was recumbent and in a stupor. Its temperature was 105° F. Blood samples were taken for testing.

Standing pools of water were seen around the stables and the house. J.G. reported seeing large numbers of mosquitoes, stable flies, and gnats congregating about the horses.

Complaining of nausea, body aches, and continual headaches, J.G. was hospitalized on July 6. Hemagglutination-inhibition antibodies to VEE were subsequently detected in his blood and J.G. became the first recorded human case of VEE in Texas. Positive neutralizing antibodies to VEE were also detected in blood from the gelding. J.G. was released from the hospital after 7 days to recuperate at home. The gelding recovered except for an impairment of its vision.

Holding Operation

The holding operation against VEE depended upon creation of an immune horse population and the control of mosquito vectors.

Systematic surveys of mosquito populations had been underway in 30 different locations since June 21. On the basis of light-trap collections, mosquitoes in the salt marsh lagoons of Boca Chica, east of Brownsville, were numerous and increasing. Populations at the other sites, however, fluctuated with new local rains and the standing water that quickly disappeared.

Information gathered by the task force indicated that approximately 35 percent of the horse population in Cameron and Hidalgo Counties had been vaccinated under the voluntary program. In the 13-county area about 10,000 horses had been vaccinated, with the greater percentage in the ranch country. This represented 4 percent of the horse population and 5 percent of the land area (254 counties) in Texas.

Low and variable numbers of mosquitoes, showing no conclusive trends, continued to be cited as justification for postponement of aerial spraying. Nevertheless, suspected VEE continued to spread. Vaccination apparently was not building an immune barrier. It was increasingly apparent that positive action beyond voluntary vaccination was needed.

Accordingly, on July 8, the Governor of Texas granted permission for State and Federal agencies to begin an aerial spray program to control mosquitoes. The priority treatment area was along the Rio Grande River from Boca Chica to Sullivan City. As rapidly as possible, treatment would be extended to include 1 million to 1.5 million acres in the 13-county area. (USDA had earlier obtained approval from the President's Council of Environmental Quality for its emergency plan to combat the introduction and spread of VEE, subject only to State acquiescence.) Other events followed rapidly.

The day after epidemic VEE was confirmed in the United States, the Texas Animal Health Commission quarantined all horses in its 13 southern counties. Four days later (July 13) USDA placed a Federal quarantine on the State of Texas to prevent the interstate spread of VEE. In effect, equines were not permitted to leave Texas unless they had been vaccinated at least 14 days prior to shipment. Concurrently, vaccine was made available throughout the State of Texas.

The quarantine, though well-intentioned and imperative in a rational disease control program, was promptly attacked by veterinarians and horse owners. The restriction of unvaccinated horses to permises, it was claimed, prevented owners from taking their horses to vaccination clinics, and was actually hampering the fight against VEE. (The shipment of horses from Mexico to the United States had been suspended on June 18.)

Two specially-equipped C123 "Provider" aircraft from the U.S. Air Force, Tactical Air Command, started spraying in the lower valley on July 10. The pesticide used was ultralow volume malathion at the rate of 3 ounces per acre. Three additional Air Force planes were added to the operation on July 13.

Meanwhile, suspect cases had started to appear outside the original 13-county area and diagnosticians were dispersed widely throughout southern Texas. By the middle of July, no one knew precisely how many horses were sick or dead. Total numbers varied from 500 to 1,000.

A crop-dusting pilot reported seeing "at least 15 dead horses" on the banks or floating in the Rio Grande River. The river was the source of drinking water for Valley cities.

Burial crews furnished by the county commissioners and city sanitation units were having difficulty keeping up with the "25-30 dead animals" being reported daily. The local rendering plant had no facilities for skinning and was reluctant to accept suspect horses. Carcasses lay rotting in the fields.

Exotic VEE had become a domestic problem of major proportions.

Expressing a common belief, the principal spokesman for the Harlingen group conceded at a press conference that the "task force had lost the battle" to prevent the entry of VEE into the United States. But, he added, it would never give up on control.

On July 16, in the face of mounting and conflicting reports of equine sickness and death extending beyond the bounds of the 13-county area of southern Texas, the Secretary of Agriculture declared that a national emergency existed from the threat of VEE. Funds from within the Department were to be used to: (1) extend the vaccination of horses, (2) enlarge the area being sprayed for mosquitoes and other vectors, (3) step up distribution of vaccine for use in the program, and (4) establish a regional emergency headquarters in Houston, Tex., to coordinate the program.

The vaccination program (with the imposition of Federal quarantines) was extended to include the States of Louisiana, Oklahoma, Arkansas, and New Mexico. In efforts to broaden and to accelerate equine protection, the vaccine was made available free of charge, with the U.S. Department of Agriculture paying veterinary practitioners a fixed fee of $4.00 per horse for their services.

Except for the maintenance of a small epidemiological study group at Harlingen, all VEE operations were transferred on July 19 to the regional headquarters at Houston.

PHASE II—EMERGENCY PLAN

The declaration of a national emergency by the Secretary of Agriculture had several salutary effects. It provided essential funds, manpower, and a legal basis for Federal action, and it galvanized State and Federal agencies into extraordinary cooperative action. On August 24, a mere 36 days after activation of the regional VEE control office, it was evident that the battle lost on the border had been won at Houston.

The regional headquarters at Houston coordinated three principal activities: vaccination, vector control, and vector and disease surveillance.

Vaccination

The last census in 1959 estimated the horse population at just under 3 million. In 1970, the estimate was 7.5 million. Of this number, Arkansas, Louisiana, New Mexico, Oklahoma, and Texas had an estimated total of 1,128,000. The reliability of local estimates, it developed, depended upon their source. In New Mexico, for example, the State recorded 24,245 horses on its rolls for the annual tax on livestock in 1970. When vaccination ended in September, 86,450 horses had been vaccinated, nor did this represent 100 percent of the population.

Despite inaccurate estimations of horse populations, Federal authorities were prepared. The Department of Agriculture on May 14, 1971, had requested the Department of Defense to set aside 1 million doses of TC-83 vaccine for emergency use in equines in the United States. Upon receipt of 800,000 doses of the vaccine on June 14, production of diluent had immediately started at the USDA's Animal Health Division Laboratory at Beltsville, Md. On July 16, an additional 1.8 million doses had been requested from the Department of Defense.

With adequate vaccine on hand, four events gave a fillip to the vaccination program: continuing spread of VEE; Federal quarantines prohibiting the movement of horses in and from Texas, New Mexico, Oklahoma, Arkansas, and Louisiana; free vaccine administered by veterinarians on a Federal fee basis; and the comprehensive public relations campaign in all the news media which carried the program virtually into every home.

Increased awareness of the disease and expanded vaccination, of course, meant a greater workload imposed upon practicing veterinarians. Regulatory veterinarians also labored under the increasing pressure of investigations and re-investigations for post-vaccinal reactions.

By July 24, approximately 53 percent of the estimated horse population had been vaccinated in Texas, New Mexico, Oklahoma, Arkansas, and Louisiana. The Veterinary Biologics Division had granted a special license to produce VEE vaccine commercially. Use of the vaccine on a Federal fee basis, contingent upon the development of State plans for acceptance, was extended to the remaining Border and Gulf Coast States: California, Arizona, Mississippi, Alabama, Florida, and Georgia.

In the following weeks, eight additional States and the District of Columbia started vaccination programs. When the program ended on December 17, a total of 2,854,191 horses (95 percent of the estimated equine population) had been vaccinated in 19 States and the District of Columbia (table 1 and fig. 3).

Based upon completed backpassage studies in horses in Mexico and field studies in the United States, the "experimental" classification of the vaccine was discontinued on August 13. Only two States, Hawaii and Massachusetts, plus Puerto Rico have not approved the use of the commercial vaccine.

Vector Control

The control of mosquito vectors was a joint operation of the U.S. Department of the Air Force, Tactical Air Command, and the Plant Protection Division, USDA. The spraying operation, which began in two C123 planes in the lower Rio Grande Valley on July 10, had covered approximately 120,000 acres in the Brownsville area by July 13.

With the declaration of a national emergency on July 16, it was decided to expand the vector control program to include the entire gulf coast of Texas in addition to the lower Rio Grande Valley—ultimately embracing a total of 22 counties—as well as Cameron and Calcasieu Parishes in Louisiana. Under the plan, the cooperating Federal departments agreed to coordinate aerial spray operations from the regional control office at Houston. Both the Air Force and the Plant Protection Division moved ahead to activate the expanded vector control program.

Table 1.—Summary of National VEE Vaccination Program

June 23 - Dec. 31, 1971

State	*Vaccination program began*	*Vaccination program completed*	*Number Fee Basis veterinarians participating*	*Number herds vaccinated*	*Number horses vaccinated*	*Estimated horse population*	*Percent horses vaccinated*
Alabama	8-17-71	9-17-71	179	53,101	122,343	129,000	95
Arizona	8-4-71	11-19-71	88	31,103	89,954	100,000	90
Arkansas	7-22-71	8-27-71	122	46,134	126,005	128,000	98
California	8-4-71	10-31-71	561	129,000	393,268	406,000	97
Delaware	9-13-71	10-1-71	25	2,364	7,916	8,000	99
Florida	8-3-71	8-27-71	241	29,274	129,215	145,000	89
Georgia	8-9-71	10-2-71	258	39,082	105,002	107,000	98
Kentucky	9-7-71	10-22-71	202	61,399	157,460	170,000	93
Louisiana	7-23-71	8-15-71	175	57,761	140,812	150,000	94
Maryland	9-7-71	10-2-71	118	13,580	44,002	48,000	92
Mississippi	8-2-71	9-10-71	132	56,940	145,099	149,000	97
New Jersey	9-7-71	10-1-71	195	10,452	30,604	31,000	99
New Mexico	7-22-71	9-15-71	84	22,853	86,450	90,000	96
North Carolina	9-9-71	10-16-71	181	56,588	119,149	129,000	92
Oklahoma	7-21-71	8-30-71	277	63,891	227,054	230,000	99
South Carolina	9-13-71	10-22-71	106	29,186	64,356	70,000	92
Tennessee	9-7-71	10-14-71	160	71,901	179,297	180,000	99
Texas	6-23-71	12-17-71	797	170,698	574,110	625,000	92
Virginia	9-15-71	10-15-71	186	44,414	111,989	118,000	95
District of Columbia	9-29-71	10-2-71		33	106	110	96
TOTALS			4,087	989,754	2,854,191	3,013,110	95

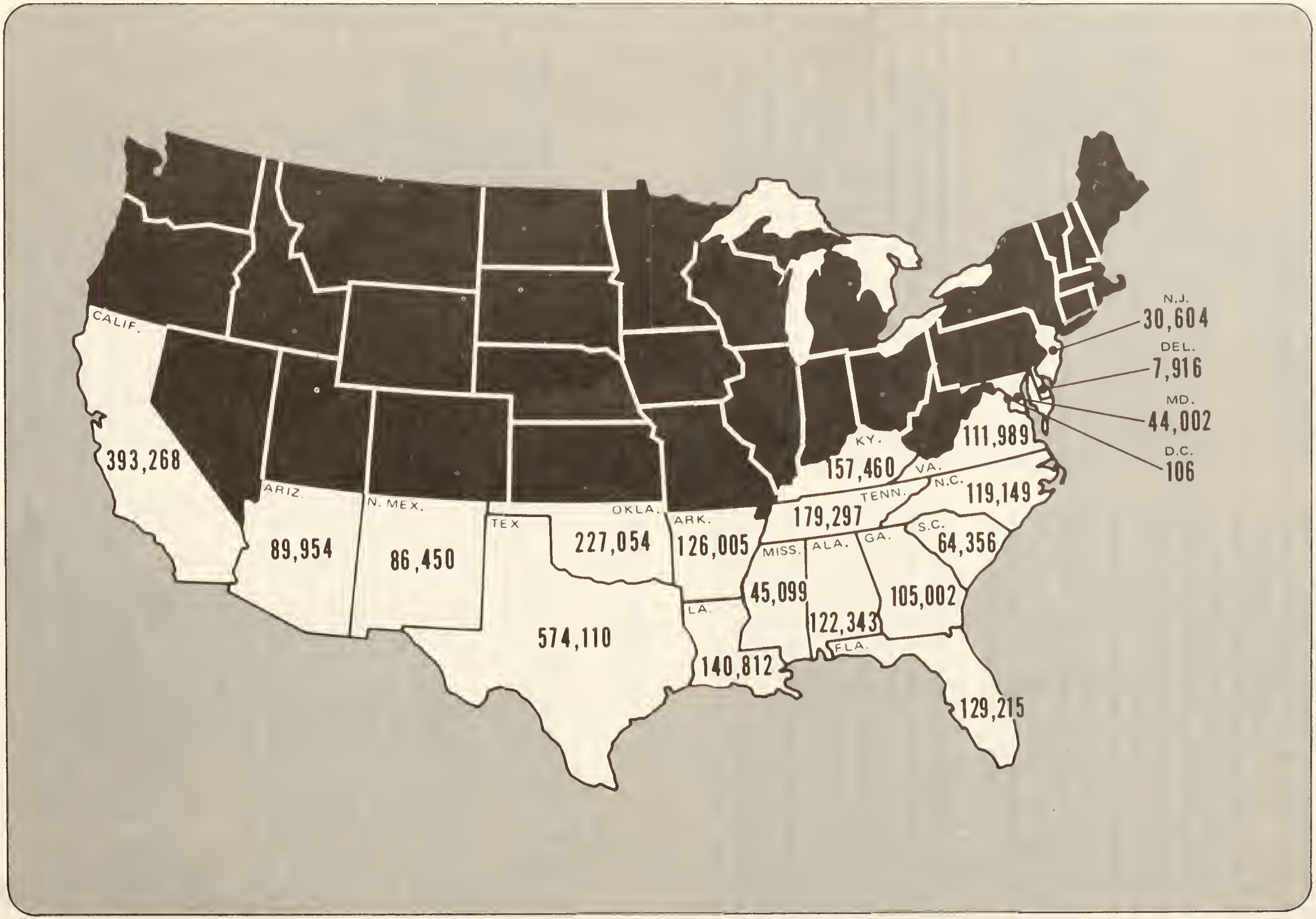

Figure 3.—VEE vaccination program, June 23 - December 31, 1971.

Under the general control of the Commander, U.S. Air Force Florida, a combat VEE Task Force was formed at Ellington AFB, Houston, to operate the ultimate fleet of nine C123 and nine C47 aircraft. The Plant Protection Division began contracting with commercial operators of multiengine aircraft, and completed support agreements with mosquito abatement units in five Texas counties. Bases of operation were established at Brownsville and Corpus Christi, Tex., and Lake Charles, La.

Civilian personnel, including 76 specialists from 18 States in the Plant Protection Division and seven specialists from four States in the Agricultural Quarantine Inspection Division were assigned to the program. Field observers and pilots assigned to each base calibrated the commercial aircraft and monitored the operations for accuracy of ground coverage.

The Air Force completed the first phase of the spray operation covering 826,000 acres in Cameron and Hidalgo Counties by July 20. With permission received from the Governors of Texas and Louisiana for expansion of the spraying program, the Air Force started Phase II the following day. The pesticides used were ultralow volume malathion at 2.6-3 fluid onces per acre and dibrom (naled), in Graham County, at 3/4 fluid ounce per acre.

Following a report on July 22 of suspected VEE in mosquitoes from Starr County, 33,329 acres were sprayed from Sullivan City to Falcon Dam. At this time the spray area extended from Lake Charles, La., through Brownsville, and along the Rio Grande River to Falcon Dam—a distance of 545 land miles.

By July 27, two additional bases had been opened at Victoria and Houston and 40 aircraft were in operation. Twelve survey teams including entomologists from the USDA's Entomology Research Division, Department of Defense, Texas A&M University, the Communicable Disease Center, and Louisiana State University under the direction of the Public Health Service were checking mosquitoes for virus activity in Texas, Louisiana, Oklahoma, and Arkansas. Mosquito populations, according to entomologists, were reduced 98 percent after spraying.

Intermittent, heavy rains from July 31 to August 8 cancelled or shortened flights. Despite delays, however, the Air Force completed its first application in the Houston area on August 7 and all planes and crews were released. The second application began at Houston the next day with contract planes only. Due to the rapidity of equine vaccinations and the effectiveness of the first aerial spraying, 12 Texas counties were eliminated from the second spraying. This diminished the need for planes and the number of contract aircraft was reduced by half.

The mosquito abatement operations were completed on August 13 and all aircraft were released. On the following day, support to the mosquito abatement districts was terminated and the vector control operations at Houston ended.

An operation of this magnitude with its mix of cooperating agencies could not fail to encounter difficulties. To commercial applicators, vector control was a business. Operators of small planes complained that all the business was going to the owners of multiengine aircraft. Owners of multiengine planes complained that the Air Force was taking business from them.

Despite a well-publicized information program, one beekeeper lost 100 colonies when he neglected to listen to the report of projected flights on the radio and took no precautions against pesticide spray.

A pilot was terminated when he dumped 50 gallons of malathion into Matagorda Bay. In its after-action report, the Air Force's Combat VEE Task Force analyzed the completed operation. These are a few of the items discussed.

Operations

At the outset, target areas were too limited and assigned too late to permit air crews to substitute alternate areas when bad weather eliminated primary targets. As a result, some sorties were aborted. The combat VEE entomologist served as liaison between the Air Force and the Department of Agriculture. He also calibrated the aircraft and performed the droplet tests to assure the correct size and dispersion of the spray. He should be a part of every future operation.

Intelligence

Crews and maintenance personnel must be informed about the hazards from prolonged exposure to malathion and dibrom, and the proper handling of pesticides and equipment. The entomologist and the flight surgeon performed this essential service.

The information staff discharged an equally important function in minimizing the disturbance from low-level flights at 150-200 feet by keeping the public and the news media informed.

Maintenance and Logistics

A single benson tank on the aircraft instead of the configuration of five 55-gallon drums for holding pesticide was suggested. This would prevent leakage and be easier to service.

Modifications of the spray systems for ultralow volume application necessitated local purchase of parts. A flyaway kit containing everything from aspirin to valves was suggested.

Communications

In areas where rain, wind, and temperature restrict operations to a few early morning hours, rapid air information is important in preparing primary and secondary targets before takeoff. The deployment of equipped, mobile teams of weathermen and radio operators to target areas was recommended for future operations.

Manpower-Personnel

Due to the early hour flights and constant exposure to pesticides, a spare crew is desirable for each type of aircraft.

Implementation of the recommendations for change contained in the report and a continuance of the practices that proved successful should ensure an effective specialized service in the event of future need.

The Combat VEE Task Force and the commercial operators sprayed 10,408,877 acres. County mosquito abatement units covered 3,086,931 acres. In sum, the vector control program treated a total of 13,495,808 acres during 35 days of operation.

Vector Surveillance

With respect to the vectors of VEE, all available forces assigned to this aspect of the program were concentrating upon determining the extent of the disease in southern Texas and controlling the vectors in the critical areas. In order to combat more effectively the still-spreading disease, it was necessary to know whether infection in mosquitoes existed outside the known epidemic area. Accordingly, by mutual agreement of the Department of Health, Education, and Welfare, and the Department of Agriculture, assistance was sought from the Department of Defense. Ten fully-equipped mosquito survey teams were requested to work in selected areas of Texas, Oklahoma, Louisiana, and Arkansas.

A force of 30 medical entomologists and preventive medicine specialists was designated under the coordination of the Entomology Consultant, Preventive Medicine Division. A base of operations was established at the Fifth United States Army Medical Laboratory, Fort Sam Houston, Tex.

Each team consisted of a medical entomologist and a preventive medicine specialist. Mosquitoes were collected at five different sites nightly, six nights each week. Three battery-operated light traps were used at each site. Collections at individual sites were repeated each week for 4 weeks.

Traps were set out in the afternoon and mosquitoes were removed the following morning. After being sorted, killed, and bottled, specimens were transported by military aircraft twice weekly in dry ice to the base for identification. Four entomologists assisted by technicians identified the mosquitoes. Each team identified and pooled an average of 3,000 specimens daily.

After identification, mosquitoes were suitably packaged, packed in dry ice, and shipped by commercial aircraft to Washington, D.C. Viruses were isolated and identified at the U.S. Army Medical Research Institute of Infectious Diseases, Ft. Detrick, Md.

Between July 28 and August 26, the duration of the vector surveillance program, the 10 teams collected 501,992 mosquitoes comprising 52 species. Of these, 36.63 percent were *Psorophora confinnis.* No VEE virus was found in any of the mosquitoes collected in the designated areas of Arkansas and Oklahoma. One isolation each was made from *Psorophora confinnis* mosquitoes collected in Louisiana and in Texas.

The isolation of VEE virus in Lousiana was made from mosquitoes collected on August 10 at Basile, Evangeline Parish. This isolate which did not kill guinea pigs by peripheral inoculation, was subsequently identified as the vaccine strain.

Epidemic VEE virus, however, was isolated from a pool of *Psorophora confinnis* collected on August 18 west of Del Rio, Val Verde County, Tex. Sick horses reported in Ciudad Acuña, Mexico, across the Rio Grande River from Del Rio, Tex., could have been a source of recycling VEE infection for mosquitoes and equines. (Epidemic VEE virus had been previously isolated from a sample collected on July 25 from a sick horse near Del Rio. The animal, a 7-year old female, had been vaccinated on July 22. It was one of 22 sick in a herd of 33 horses. According to a ranch employee, the mare showed no signs of sickness when last seen at noon on July 24. The mare was found dead the next morning.)

The methods employed in the vector surveillance program, it was concluded, would have detected the spread of epidemic VEE in mosquitoes in Louisiana, Oklahoma, and Arkansas during August 1971. The detection of epidemic VEE virus in mosquitoes near Del Rio, Tex., on August 18, confirmed that the only notable spread of infection in the surveillance area occurred along the Rio Grande River. The isolation of epidemic VEE from a horse at Presidio on September 11, 200 miles northwest of Del Rio, presents two alternatives: surveillance did not last long enough nor extend far enough, or other means of dissemination and spread were involved. The latter possibility is discussed at greater length in the section entitled Maintenance and Spread beginning on page 21.

The regional VEE headquarters was transferred from Houston to the Ready Room at Beltsville, Md., on August 24. Although there was little doubt that epidemic VEE had entered the United States through the Lower Rio Grande Valley of Texas, many questions were still unanswered. For example, when and in what manner had the disease been introduced? By what means was the disease so speedily and widely dispersed?

Proof of specific cause and effect are not readily established in biological phenomena. The alternative is a reasonable interpretation of the most probable associations and causal relations. These all catalyzed in the Lower Rio Grande Valley in the late spring and summer of 1971. First, the environment.

Environment

The Region

Two forces have shaped the destiny of South Texas for as long as there is written record: droughts and floods.

The first settlements along the Rio Grande were at Santa Ana de Camargo and Villa de Reynosa in 1749. Reynosa comprised 533 persons of whom 310 were Indians, and 6,000 head of stock. The early settlers tried to take advantage of the extreme soil fertility and the readily available water for irrigation. Intermittent droughts and floods, however brought failure to irrigation cropping. Efforts then turned to ranching, and by the middle of the 19th century the cattle industry dominated the economy.

Since the operation of the cattle industry depended upon horses, hunting and trapping mustangs, the wild descendants of the stock brought in by the Spanish explorers, became an important enterprise.

Mosquitoes and disease are also linked to the history of southern Texas. In the mid-1850's the stage took 4 days and nights to travel from Corpus Christi to Brownsville. The journey was through sparsely settled country and passengers were obliged to bring their own provisions. Large numbers of wild, unclaimed cattle and horses roamed the inhospitable sandhills and salt grass prairie. Frequently during the journey, the driver would cry, "Curtains down!" and the passengers would suffer near-suffocation in the stage while a sandstorm whirled outside. At other times the cry, "Curtains down—and tight!" was the sharp warning for passengers to protect themselves from the clouds of mosquitoes that swarmed over horses and humans. Frequently these attacks caused fever.

Early in the 20th century, the first railroad pushed into the Valley. Rail service provided a means of quickly moving agricultural produce to market. This made feasible large irrigation projects that once again transformed the region.

Geography

The Lower Rio Grande Valley, in point of fact, is not a valley at all. The land slopes northeast away from the Rio Grande. This permits gravity flow of irrigated water from the river making the area a delta rather than a valley.

The river originates in San Juan County, Colo., and empties into the Gulf of Mexico several miles southeast of Brownsville. It is 1,885 miles long.

The largest natural drainage channel in the system of surface waters is the Arroyo Colorado. It runs easterly from Mercedes to Harlingen emptying into the Laguna Madre. Its maximum width is 200 feet and its maximum depth 30-40 feet.

South of the arroyo the land is broken by resacas, former channels of the Rio Grande. Through the years, as the river has worked south, these depressions, closed at both ends, have been left behind. Periodically they are filled with water pumped in from the Rio Grande and serve as reservoirs for irrigation purposes.

An artificial floodway system has been constructed under sponsorship of the International Boundary and Water Commission. It includes two major channels which carry off floodwater and drainage water from adjoining land. The main floodway, 30 miles long, extends from Mission to the Arroyo Colorado. The north floodway extends northeasterly, about 50 miles from the juncture of the main floodway and the arroyo to the coastal zone.

Underground water is found at 0-3 feet on 2 percent of the land in the Valley; at 5-10 feet on 65 percent of the land; and at a depth over 10 feet on 14 percent of the land. Flow of groundwater is from west to east. The rate of flow is slow due to the flatness of the land and the fine texture of the soil.

The Valley comprises two principal types of country: the alluvial lands south of the Arroyo Colorado and the upland plains of the Coastal Prairie. The alluvial region extends 20 miles north of the Rio Grande. These sandy to clay soils are fine textured delta deposits from the Rio Grande. The soils are fertile except toward the coast where heavy clay causes poor drainage and where there is high salt content.

Just north of the alluvial soil, phasing into the Coastal Prairie, is a belt of darker-colored, principally loamy soils that retain moisture. This belt extends from southwestern Hidalgo County to the coast and includes Cameron and Willacy Counties. Still farther north on the upland plain are the light-colored, sandy loam soils of the Coastal Prairie.

Settlement of the region displaced the early grassland. Woody vegetation rapidly took over. In the western deserts, mesquite is a shrub. In the humid, valley climate, mesquite is a tree. Mesquite, chapparral, prickly pear, guajillo, ebony, ratama, tornillo, huisache, and other trees and brush form a dense, tangled, and thorny jungle. After rain, grasses, flowers, and weeds of all kinds spring up. Along water courses cottonwood, willow, and hackberry are found. Along the coast the vegetation is mainly marsh and coastal weeds and grasses. Interspersed among the salt flats are low vegetated ridges supporting thick thorny shrubs and brush. In the alluvial region, cotton, grain sorghum, vegetables, and citrus are grown under irrigation. North of the alluvial region the same crops are dry farmed. Large areas are used only for ranching.

The Valley is honeycombed with surface water in its coastal marshes, arroyos, resacas, drainage canals, and irrigation ditches. Fluctuating water levels frequently leave extensive exposed areas of standing water and vegetation. (Resaca in American-Spanish also means "mud and slime".) These water courses, the myriad puddles that form in the caliche-clay soil, and the extensive areas of brush along waterways and even on cultivated acreages—all provide an optimal environment for the development of mosquitoes and biting flies.

Weather and Climate

The climate of the Lower Rio Grande Valley is partly man-made. It is semiarid due to lack of rainfall. It is semitropical and man-made due to the extensive irrigation. Temperatures throughout the year average 74° F., ranging from 51° F. in January to 97° F. in August. The average date of the first killing frost is December 15; the last killing frost occurs on January 23.

Hail, confined to Hidalgo County, falls every year Traces of snow, too small to measure. have appeared several times. On February 14-15, 1895, however, 6 inches of snow blanketed Brownsville. Sunshine each year averages 2,799 hours. The average growing season is 326 days in the Brownsville area and somewhat shorter in the west.

Normal rainfall ranges from more than 30 inches at Brownsville to less than 20 inches at Mission. Normally the greatest rainfall occurs in May and June, and in September and October. Most of the summer rainfall comes in the form of thunderstorms and often a single storm will provide the entire month's rainfall.

The greatest amount of rain to fall in any 24-hour period was 11.91 inches during September 1866. The longest period of rainfall, however, occurred in 1882. All of Cameron and part of Hidalgo were inundated from the first part of August until mid-November. Mosquitoes flourished and yellow fever spread through the human community. Night and day, it was reported, the carpenter's hammer was heard driving nails into new coffins, and day after day the dead were carried by boat to the cemetery.

Thirty-two inches of rainfall joined the constantly rising river. Floodwaters extended from the center of Brownsville 46 miles north to the Sauz ranch in the northeast corner of Willacy County.

Generally, the prevailing winds in the Valley are from the southeast 10 months of the year. During December and January the prevailing winds usually are from the northwest. The highest wind velocity recorded was 80 miles an hour, on September 5, 1933. The area is subject to tropical storms.

Table 2 gives temperature, precipitation, and wind data for the Brownsville, Tex., area, February-July 1971. Table 3 compares the precipitation rate of various areas for June 1971.

Table 2.—Climatological data, Brownsville area, 1971

Month	*Temp. (average)*	*Precipitation (total inches)*	*Wind velocity (m.p.h.)*	*Wind direction*
February	67.2	0.30	13.2	N
March.........	72.1	Trace	14.7	N
April	74.3	1.82	13.7	SE
May	79.5	2.47	14.1	S
June	82.8	3.44	12.7	SE
July	83.4	1.08	11.7	SE

Table 3.–Comparative precipitation in various areas, June 1971

Place	*Precipitation (total inches)*
Brownsville	3.44
Harlingen	2.33
Raymondville	2.68
Weslaco	3.30
Edinburg	2.36
McAllen	5.90
Mission	3.86
Rio Grande City	1.13

MAINTENANCE AND SPREAD

Two principal measures were employed to contain VEE in northeastern Mexico, vaccination and mosquito control. Yet VEE appeared in the United States. Subsequent attempts to limit its spread from a small focus in southeastern Texas by the same methods also failed.

In retrospect both efforts failed largely for the same reason: control measures were applied after the disease had become established and then were inadequate for the threat. But the problem is more complex than that simple declaration indicates, for there is still less than complete understanding of the complex interactions involved in the maintenance and spread of the disease.

For a long time a mosquito-vertebrate cycle was believed to be the principal means for maintaining and disseminating VEE virus. The prevailing cycle at any particular time depended upon the vectors and the available hosts and their immunological status. Simply stated, infected vectors passed from endemic or epidemic regions to other areas where a small number of immunes provoked epidemics. Once established, the virus might persist for months or years. Ultimately, unfavorable seasons or lack of susceptible hosts caused epidemic virus to disappear from the area until a subsequent invasion by new seed virus.

Closer study of the natural history of VEE, however, reveals that its maintenance and spread is more involved than a simple rodent-mosquito-large host transfer.

Intimations of the complexity of VEE epidemiology came after the Colombian outbreak in 1967. It became evident that VEE was not limited, as had been thought, to small foci in a rodent-mosquito cycle—periodically spilling out into equines every 10 or 20 years and then retreating to its endemic source for another decade or two. Contrary evidence indicated that VEE existed not only beyond its known endemic foci, but remained active when there no longer were susceptible equines for its maintenance.

VEE virus was isolated from spiny rats in the foothills of Colombia, from tree climbing rats and squirrels at elevations of 8,900 feet, and from rats on the Atlantic coast 1 year after the epidemic ended *(26).*

In 1970, the epidemic in Colombia appeared in different areas from those infected in 1967-68. At the headwaters of the Magdalena River the epidemic progressed slowly among equines and did not affect humans or other domestic animals. Large populations of cotton rats in the precise locations with horses were not involved.

Cotton rats in nature frequently have been found infected with VEE virus, presumably from mosquito bites. But under laboratory conditions cotton rats have been infected by intra-nasal or intra-ocular instillation of virus in cotton rat urine or feces *(43).* Thus, contact spread via urine, feces, or the nasopharynx may provide another method for the maintenance and spread of virus in this small mammal population.

Man and horses, likewise, have been infected via the nasopharynx, with horses developing viruria. In laboratory studies, horse-to-horse transmission of VEE was successful when *Aedes triseriatus* mosquitoes were used as the transmitting vector. Virus was shed in the nasal, ocular, and oral secretions, as well as in the urine and milk of an infected horse. VEE was also transmitted by *A. triseriatus* mosquito to dogs *(9)* and to wild birds.

Field studies conducted in Central America by investigators from MARU and the Cornell University Medical College from 1965 to 1969 substantiated the complex natural history of VEE. They added information about new hosts and their interactions with VEE viruses. They also confirmed the coexistence of endemic and epidemic areas and the likelihood that several cycles of virus transmission occur, some or all of which may operate in the same area at the same time.

In sum, then, multiple mechanisms and many life forms and agencies may be equally as important as the mosquito-vertebrate cycle in local transmission as well as in the broader geographic maintenance and spread of VEE.

Vaccination

More than 2 million doses of TC-83 vaccine had been used to immunize equines in Latin America from 1967 to 1970. During this experience horse deaths reportedly ended 7 to 10 days after vaccination. There was evidence that vaccination protected 90 percent of equine populations. Following vaccination of all equines in rural communities, human illness also ceased.

Extensive vaccination of equines in epidemic areas, however, did not always provide an impenetrable barrier to the spread of the disease, nor prevent a recurrence of epidemics in the same areas. In endemic areas, on the other hand, unvaccinated equines seemed to be protected against infection by the epidemic types of virus.

The failure of TC-83 vaccine to contain VEE both in northeastern Mexico and in southern Texas is not a judgment against the vaccine. In Mexico, a variety of circumstances prevented the creation of a realistic, timely barrier against VEE. The program started rapidly with owners being instructed to bring their equines to designated vaccination centers. In the first 4 days, 14,000 equines were vaccinated at centers in Reynosa, Matamoros, Valle Hermosa, and San Fernando. Then the program faltered.

Due to poor communication facilities, residents of some ranches and communities were not notified. Prolonged drought conditions in most areas deterred many owners from subjecting their animals to the additional stress of travel to vaccination centers. Census estimates of equine numbers were inaccurate.

After a reported drought of several years duration, heavy rains started shortly after the campaign began. Owners then complained that with the flooding of fields their animals would not come to the waterholes to be rounded up for vaccination. The vehicles provided to the vaccination teams had limited capability on poor roads, and vaccinators were unable to reach many communities. Observers estimated that approximately 25 percent of the equine population was vaccinated. Vaccination teams subsequently returned three and four times to obtain maximum coverage.

The rains increased populations of vector mosquitoes thus accelerating and enlarging the area of infection. As the disease spread inland from the coast, attention and funds were diverted from the vaccination program on the international border. Massive die-offs among both vaccinated and unvaccinated equines continued through June and July. The purported barrier was a sieve.

In the Lower Rio Grande Valley, vaccine was administered in the face of an approaching epidemic, or, more to the point, of a disease that was probably already present but undetected.

Voluntary vaccination programs do not always assure adequate population protection. The necessity of paying a fee for veterinary services further diminished public acceptance. Some horse owners were unable to pay, while others who are able, refused to pay for what they believed should be provided free. The tendency was to protect the more valuable horses, leaving from 50 to 65 percent of the population unprotected during the first crucial 3 or 4 weeks.

Still other factors frustrated building an immune barrier. In some instances, despite warnings to the contrary, horses were vaccinated when they were visibly sick. In other cases, apparently normal horses living in infected surroundings died with clinical VEE after being vaccinated. It is assumed the vaccine enhanced an incubative infection. Horses that were vaccinated during the febrile period became sick rapidly, and many died within 24-48 hours. Where death did not occur recovery took several weeks.

Certain actions of overzealous veterinary practitioners may also have abetted the spread of VEE in southern Texas. This recital concerns only three verified examples which were witnessed and reported by either ranchers or other veterinarians.

Immediately upon release of the vaccine in June, a practitioner from the Brownsville area flew to San Antonio to vaccinate two herds of 80 horses. (The practitioner owned one herd of 72 horses, the second herd of 8 was owned by the practitioner and another individual.) This procedure was in violation of the agreement by practitioners to limit vaccination to the 13 southern counties.

There was a great urgency to protect as many horses as rapidly as possible. In that spirit, a man and wife veterinary team stated that they had started vaccinating one day at 9:00 a.m. and had continued until midnight. This was probably a record. But in other similar large scale operations, observant ranchers noted that veterinarians continued to use the same unsterilized needle. Epidemic VEE was confirmed on one of the ranches where this practice was observed. (Perhaps to counteract this temptation, when the State of Virginia started its vaccination program, vaccine was dispensed to veterinarians in its northern counties each day by an employee of the State Department of Agriculture. The employee counted the disposable needle-syringes used the previous day and compared this with the vaccine distributed to assure that no needle and syringe had been used more than once.)

The third act was an admission by a veterinary practitioner that after he had reconstituted vaccine for use it had been frozen, thawed, and refrozen for use 2-3 days later. Instructions recommended use of vaccine within 12 hours of its reconstitution.

No attempt was made to document irregularities. It is not suggested either that these particular acts were typical or that they precipitated the widespread epidemic in Texas. They are noted solely because they are components in the epidemiology of an epidemic about which much is uncertain and which may be understood only from an understanding of all the facts, including the interrelationship of many small, seemingly unimportant parts.

Assuming, for the sake of developing hypotheses, that VEE was in the United States before vaccination began in the Valley, how did it enter and how did it spread?

Viruses

Arboviruses apparently have evolved several systems of survival. One is believed to be a balanced low-level relationship with regular hosts, possibly including insect vectors, causing minimal damage to the host. Another system for virus survival is by epidemic spread among non-immune alternate hosts aided by insect vectors.

The first system, which preserves the host, favors virus survival. The second system, since it either destroys the host or renders it immune is less favorable to virus survival. In effect, then, epidemics are self-limiting even though there may be extension in range of virus activity *(25)*.

An additional consideration in arbovirus ecology specifically related to VEE virus is the evidence that certain strains of the virus do not change their silent role. This is believed true of a strain found in Latin America as well as of the strain identified in Florida. The relatively mild strain common in the lower Gulf Coast area of Mexico seldom causes disease in equines or in man. In fact, infection with this mild strain appears to confer immunity to the virulent epidemic form of VEE virus.

The strain identified in south Florida has never been recorded in epidemic form. Antibodies to three different strains of VEE virus, however, were found in a large percentage of Indian residents near the Florida Everglades. A virus apparently identical to that of VEE was also isolated from pools of *Culex* mosquitoes, subgenus *Melanoconion*, collected in the Everglades. Concurrently, specific antibodies to VEE virus were found in cotton mice and cotton rats caught at the same site in the Everglades. Birds were essentially antibody negative. Low rates of antibody were found in deer, bobcats, and raccoons to the north of the endemic zone.

Under experimental conditions the Florida strain fails to develop sufficient levels in the blood of horses to infect mosquitoes *(37)*. Nevertheless, the Florida strain seems to have a neurotropic potential for horses though its effects are more benign than other strains of virus *(18)*. Despite knowledge since 1963 of the presence of VEE virus in Florida and serological evidence of equine infection, no equine disease has been seen *(14)*.

Two coexistent phenomena seem to be present among arboviruses in nature. In one case, certain strains of VEE virus, although detectable by laboratory methods, neither revert to a virulent form nor cause frank disease. In the second case, other strains of virus under the stimulus of favorable conditions of environment, biology, or climate may change from a nonvirulent, silent status to a virulent epidemic form.

It is postulated that the epidemic strain of VEE virus is preceded by a latent form which may be present in a host for several weeks or longer, though undetectable by laboratory procedures. Malnutrition, parasitism, and subhealth in the host may help trigger the latent virus into the recognizable epidemic form. During this transition equines may develop high levels of viremia before clinical signs appear thus acting as an unsuspected source for spread of the virus. (Moreover, different types of VEE virus may evoke different host responses.) The potential interval of latency plus approximately 2 weeks for incubation of the virus could partially explain the almost simultaneous emergence of epidemic VEE in distant geographical areas, as well as its appearance when mosquito populations are minimal. At the same time, transovarial passage of the virus, experimentally demonstrated with the eggs of *Mansonia perturbans* mosquitoes *(3)*, could provide a means for prolonged survival of virus.

The types of VEE virus, the regions in which they have been found, and some of their characteristics are summarized in figure 4 and in table 4.

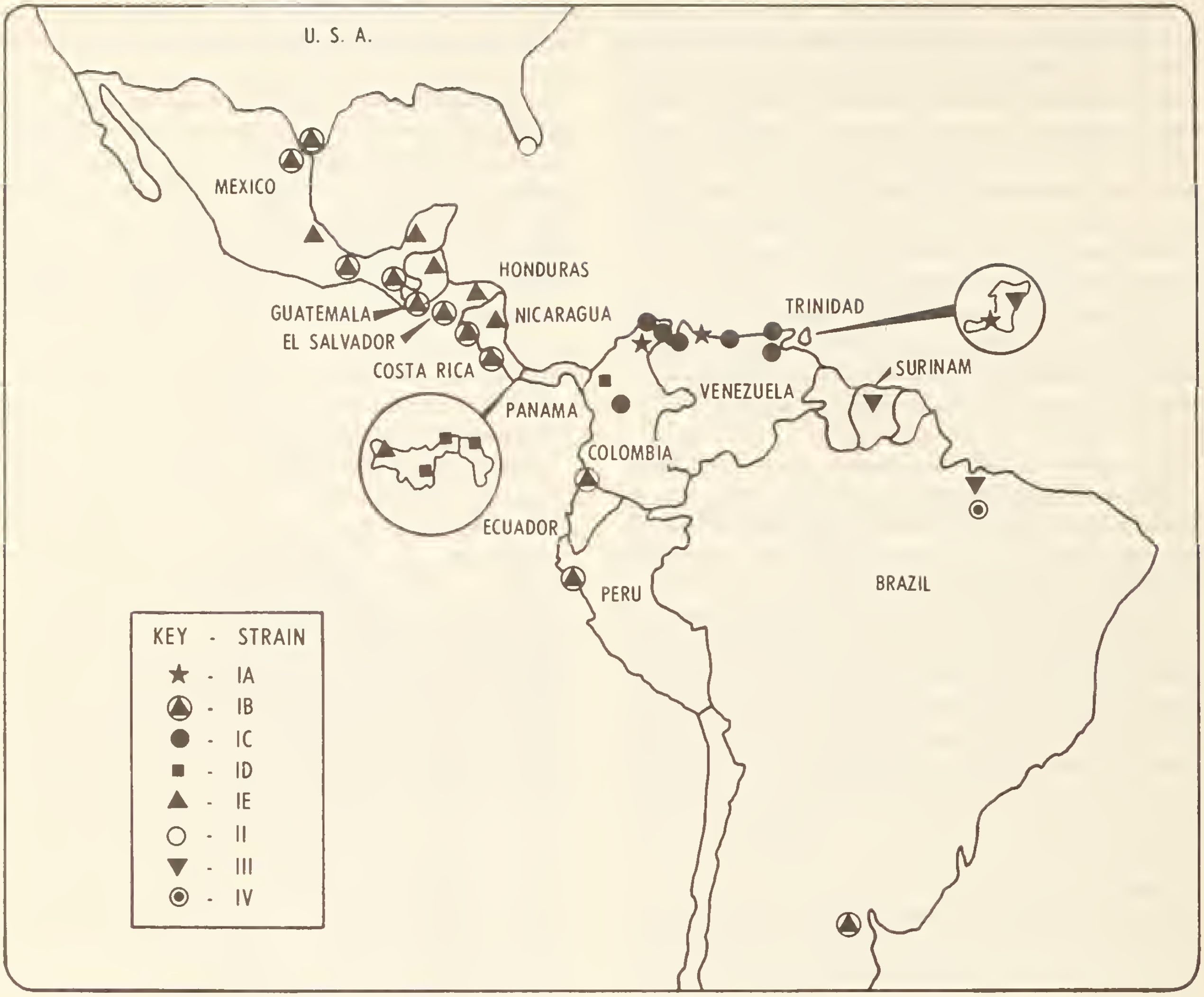

Figure 4.–Geographical distribution of VEE variant strains. Adapted from *(42)*, *(36)*.

Table 4.—Viruses of Venezuelan Equine Encephalomyelitis: Types, Subtypes, Locations, and Characteristics

Type	*Subtype*	*Location*	*Characteristics*
I	IA (epidemic)	Colombia, Venezuela, Trinidad[1]	These are epidemic strains highly virulent for equines which produce viremia before signs appear. The blood of the host probably is infectious for most species of mosquitoes. As viremia disappears, virus neutralizing antibodies begin to circulate and the more severe clinical signs appear.
	1B (epidemic)	Ecuador, Peru, Argentina, Central America, Mexico, and Texas	
	1C (epidemic)	Venezuela	
	1D (endemic)	Colombia and Panama	These endemic strains produce disease in humans and in some rodents. Infection in equines is without clinical signs. Infection produces neutralizing antibodies that protect equines against the epidemic viruses. The endemic types are found in moist, tropical rain forests and jungles. They are involved in a rodent-mosquito-rodent cycle.
	1E (endemic)	Western Panama, and the Caribbean Coasts of Nicaragua, British Honduras, Guatemala, and Mexico. Also the Panamerican Isthmus, through the Darien Jungle and into Colombia	
II	(endemic)	Florida	These are two distinct types, non-pathogenic for equines.
III	(Pixuna)	Brazil	
IV	(Mucambo) (Bush Bush)	Brazil Trinidad[1]	This type includes two strains serologically identical, also non-pathogenic for equines.

[1] Both endemic and epidemic virus cycles exist in Trinidad.

Vectors

Mosquitoes

Mosquitoes were first incriminated as vectors of VEE during the Trinidad outbreak of 1943 *(16).* Of the mosquitoes caught in stable traps in the area of the disease, 90 percent were *Mansonia titillans.* When VEE virus was isolated from some of these, *M. titillans* was considered to be the prime vector. An attempt was made to reproduce the disease under laboratory conditions. Infected female mosquitoes, it was found, would not feed upon small animals. Nevertheless, 50 percent of the same mosquitoes willingly fed upon a donkey, producing a fatal infection.

Despite the affinity of *M. titillans* for feeding on equines, the species does not range far from its breeding ground. *Aedes taeniorrhynchus*, a long-range flier and an abundant species in Venezuela, was credited with infecting the first case in Trinidad.

Since 1943, numerous investigators have implicated various species of mosquitoes as actual or potential vectors wherever endemic and epidemic VEE has been detected. The species from which virus has been isolated appear in table 5.

In view of this evidence of a mosquito-virus association, monitoring mosquito populations near the United States-Mexico border became one of the first preemergency measures undertaken. Its purpose was to determine population trends, identify species of mosquitoes, and attempt virus isolations from trapped mosquitoes. In point of fact, a study of mosquito dynamics became the primary alert to signal the start of the mosquito control program.

At the outset both larval and adult populations were surveyed. By the end of June, with the disappearance of standing water from recent rains, larval surveys were discontinued. Adult mosquitoes continued to be surveyed by means of battery-operated light traps placed in different locations in the Rio Grande Valley. Traps were checked at intervals of 4-7 days.

By July 2, 13 of 24 traps showed increases in mosquito numbers over the previous inspection. In the view of the evaluators, however, diminishing standing water and variable mosquito numbers at the different sites prevented a determination of overall trends. The numbers were considered insufficient to justify the start of aerial spraying.

On July 8, the traps in the salt water marshes of Boca Chica had increased 13 percent in 2 weeks. Aerial spraying was begun on July 10.

Meanwhile, (although it was not confirmed until late in July) *Psorophora confinnis* mosquitoes infected with VEE virus were already in the United States. Epidemic

Table 5.—Mosquitoes from which Venezuelan Equine Encephalomyelitis virus has been isolated, 1943-1972[1]

Species	Location	Year
Aedes (Ochlerotatus) angustivittatus	Panama	1959-62
Aedes atlanticus tormentor	Florida	1963-69
Aedes scapularis	Venezuela	1962-64
Do	Mexico	1963-64
Aedes serratus	Ecuador	1952
	Brazil	1960
	Trinidad	1960
	Venezuela	1962-64
Aedes sexilineatus	Colombia	1967
Aedes sollicitans	Texas	1971
Aedes taeniorrhynchus	Venezuela	1954
	- - - - do- - - -	1962-64
	Florida	1963-69
	Guatemala	1969
	Texas	1971
Aedes thelcter	- - - - do- - - -	1971
Anopheles aquasalis	Venezuela	1962-64
Anopheles crucians	Florida	1963-69
	Texas	1971
Anopheles neomaculipalpus	Trinidad	1944
Anopheles nimbus	Brazil	1963
Anopheles pseudopunctipennis	Texas	1971
Anophelini species	Panama	1959-62
Culex coronator	Mexico	1963-64
Culex (Melanoconion) sp.	Brazil	1960
	Florida	1964
	Guatemala	1969
	Texas	1971
	Mexico	1971
Culex (Melanoconion) aikenii	Panama	1970
Culex (Melanoconion) iolambis	Mexico	1963-64
Culex (Melanoconion) opisthopus	- - - - do- - - -	1963-64
Culex (Melanoconion) taeniopus	Panama	1959-62
Culex (Melanoconion) vomerifer	- - - - do- - - -	1959-62
Culex mojuensis	Brazil	1961
Culex nigripalpus	Guatemala	1969
Do	Florida	1963-69
Culex pipiens quinquefasciatus	Panama	1959-62
Culex tarsalis	Mexico	1971
Culex thriambus	- - - - do- - - -	1963-64
Culex (Tinolestes) sp.	Brazil	1961
Culex sp.	Trinidad	1960
	Mexico	1963-67
Deinocerites pseudes	- - - - do- - - -	1963-64
	Costa Rica	1969
Haemagogus mesodentatus	Texas	1963-67
	Mexico	1963-64
Haemagogus sp.	Brazil	1960
	Texas	1971
Mansonia titillans	Trinidad	1943
	Guatemala	1969
Psorophora albipes	Colombia	1961
Psorophora ciliata	Texas	1971
Psorophora cilipes	Guatemala	1969
Psorophora confinnis	Venezuela	1962-64
	Guatemala	1969
	Texas	1971
Psorophora cyanescens	Mexico	1971
Psorophora discolor	Texas	1971
Psorophora ferox	Trinidad	1960
	Brazil	1961
	Colombia	1961
Sabethes sp.	Brazil	1960
Wyeomyia mitchelli	Mexico	1963-64
Wyeomyia sp.	Colombia	1967

See footnote at end of table.

[1] For detailed references see *(34)*, *(31)*, *(17)*, and *(15)*.

VEE had infected horses in Cameron County and had spread 200 miles to the north.

This succession of events posed queries concerning the reliability of light traps in estimating mosquito populations in predicting disease potential. It also raised the possibility that other agents were playing more than a minor role in the spread of the disease.

Mosquito light traps are highly specific. Since they sample only immediate localities, they do not provide a reliable measure of total population numbers. Buildups can occur in other areas before traps are installed. This was well illustrated when 65,000 mosquitoes were caught in five traps in one night at Ejido Palo Blanco, Mexico, while zero mosquitoes were caught 7 miles away on the United States side of the Rio Grande River.

The traps were also an unreliable barometer of disease potential. VEE virus was isolated from *Psorophora confinnis* mosquitoes collected near San Pedro, Tex., on June 28. Concurrently, the traps near San Pedro, were one of 10 locations showing a decrease (62 percent) in mosquitoes collected between the first inspection on June 23 and the second on June 28.

Also to the point was the subsequent confirmation of epidemic VEE on premises (like the first case on the D.P. premises) where residents were being "eaten up" by mosquitoes while nearby traps attracted insignificant numbers.

The placement of traps can affect both the samples and the judgments that are made from them. Traps placed near breeding areas project newly-emerged populations with the objective of control by spraying. This procedure does not, however, adequately sample populations that are actively transmitting disease, that is, mosquitoes taking their third or fourth blood meal. From observation of the epidemic that suddenly emerged in many localities in southeastern Texas, infected mosquitoes flying over from Mexico probably were already at this stage of transmission.

VEE virus was isolated from pools of three species of mosquitoes collected at Ejido Palo Blanco, Mexico, on July 3. It will be recalled that clinical VEE (subsequently confirmed by virus isolation) was first noted among equines in the community on June 25. Another factor that limits the reliability of light traps is the biology of mosquitoes expressed in their flight behavior. Instinctive flight behavior might also explain why VEE could appear almost simultaneously in many distant locations.

Flight of Mosquitoes.—Mosquitoes have two types of flight—migratory and appetitive. The former is adaptive and purposive. It occurs as a synchronized mass flight soon after emergence, dispersing populations from breeding to resting sites. Appetitive flights are searches for food that usually occur after (but may occur during) migration.

In migratory, or adaptive, flight mosquitoes rise above their environment ensuring displacement on the wind; they are undistracted by common objects of orientation; and the flights are longer than those by the same individual or species on other occasions.

The migratory flight of *Aedes taeniorrhynchus* was studied in Georgia and in Florida. It was found that when mosquitoes emerged they flew upward at a 30°-60° angle undistracted by a light trap or bait traps. In one study, of 1.5 million marked mosquitoes that were released, only 35 were caught in a light trap 2 to 3 days after emergence. In another study, when 2 million were released, 428 were recaptured 2 to 5 days later at distances 4 to 21 miles from the source of emergence *(19)*.

In Florida, wind accounts for the regular dispersal of *Aedes taeniorrhynchus* from 30 to 60 miles. *Aedes sollicitans* has made journeys up to 28 miles across water and the same species has been found 110 miles offshore.

The migratory flight of mosquitoes aided by prevailing winds from the southeast, depicted in figure 5, undoubtedly helped the northward spread of VEE to the Rio Grande River. Short-range feeding flights of the abundant mosquito populations in northern Mexico may have initiated the first cases in the lower valley. Migratory flights of new broods from the Rio Grande River area and Boca Chica, some of which had fed and become infected, (again assisted by prevailing winds from the southeast) could then have helped disperse VEE through southern Texas, perhaps even as far as Three Rivers. Enormous numbers of mosquitoes can be infected by a single viremic horse.

During the epidemic in Texas, CDC, Atlanta, Ga., isolated VEE virus from pools of 12 species of mosquitoes. Approximately 98 percent of the virus isolations were made from *Aedes sollicitans.* One VEE virus isolate was obtained by investigators of the Walter Reed Army Institute of Research, Washington, D.C., from a pool of *Psorophora confinnis* mosquitoes.

Aedes sollicitans is the common salt marsh mosquito and the most important of the species along the Atlantic and Gulf coasts. Eggs are laid in potholes or depressions as well as in any moist level place in the marshes or adjacent grassy areas. Eggs can withstand long periods of drying. Rains or seasonal flooding produce hatching of large broods. The females are fierce biters and strong fliers. Large swarms commonly migrate just before dark. Flights of 40 or 50 miles inland from the coasts are not uncommon. The mosquitoes settle in the grass and bite animals and humans even in full sunlight *(21)*.

Psorophora confinnis is closely related to *Aedes.* The eggs withstand drying and may lie dormant for long periods. Rains and flooding stimulate hatching. The larvae develop rapidly and may complete their metamorphosis in small temporary pools of water. The females are fierce biters and the species is an important economic pest.

The species of mosquitoes from which VEE virus was isolated and the location and dates of collection are given in table 6.

Biting Flies

Mosquitoes are not the only insects that travel great distances on the wind. *Simuliidae*, black flies, also use this as a normal method of rapid dispersal soon after emergence. This is particularly true of unfed but fertilized females. Males tend to remain near the emergence site. In various parts of the world large swarms of *Simulium* commonly have been reported 50-90 miles (and in some cases 140 miles) from their breeding sources near streams and waterways. These flights may be made at heights of 1,000-5,000 feet. *Simuliidae* may be important vectors particularly at higher altitudes above the usual range of mosquito flight. The adults attack all warmblooded animals. During the epidemic in Colombia in 1967, 11 VEE virus isolations were made from five species of *Simulium (39, 26)*.

Culicoides likewise attack warmblooded animals, though the distances they travel in migration are not as well documented. Reports of flights 4-5 miles over mountains 1,200 feet high seem to be exceptional. Still, some *Culicoides* reportedly fly in winds up to 9 miles per hour. There are only two records of the study of *Culicoides* in relation to VEE epidemics. The first was during the outbreaks of VEE in Venezuela from 1962 to

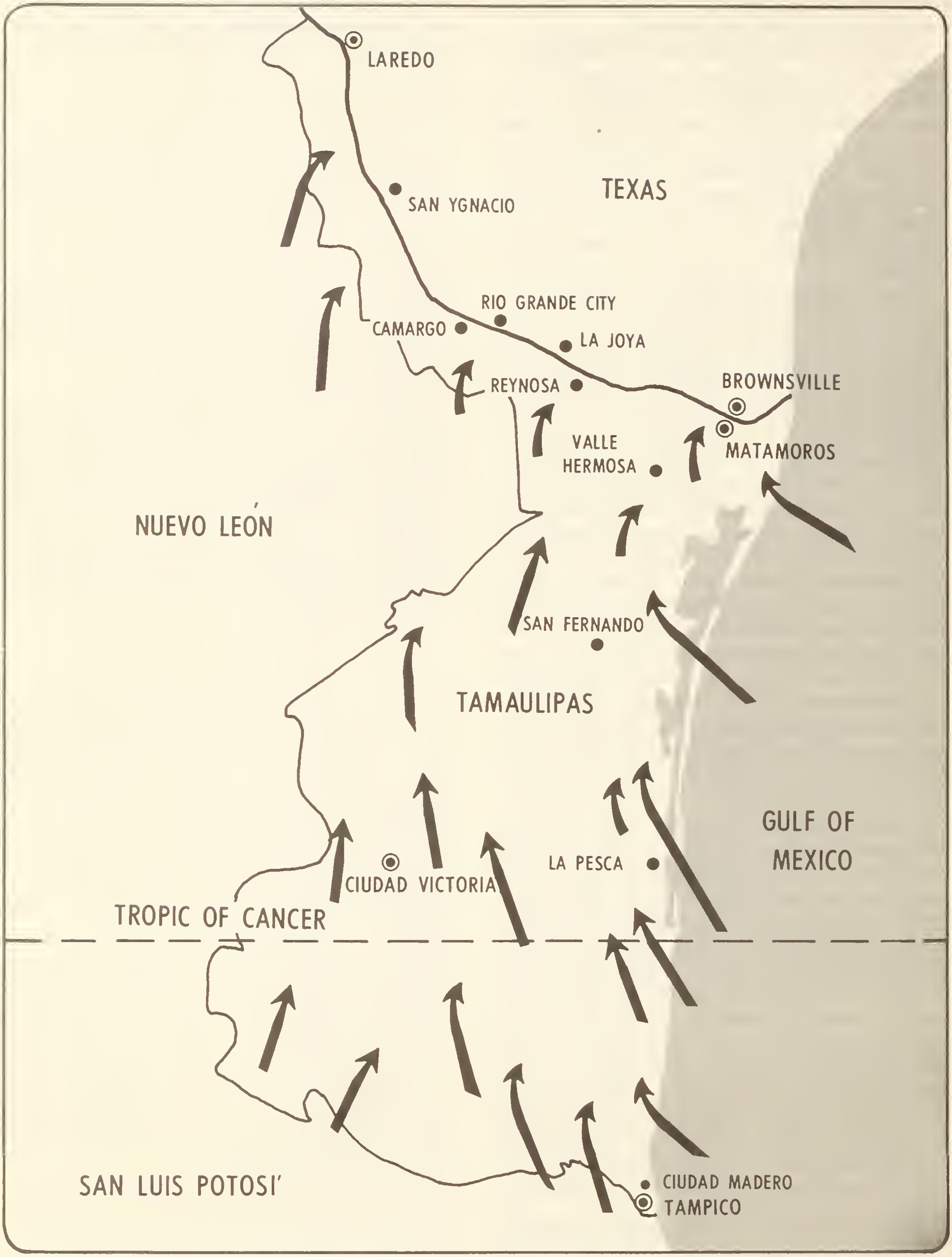

Figure 5.—Direction of the dominant summer winds in northeastern Mexico and southern Texas.

Table 6.—Venezuelan Equine Encephalomyelitis Virus isolated from pools of mosquitoes collected in South Texas, confirmed by complement-fixation tests, June 28-August 18, 1971

Location	Date collected	Species
San Pedro, Site 12	June 28	*Psorophora confinnis*
Ejido Palo Blanco, Mexico	July 3	*Aedes thelcter* *Culex (Melanoconion)* sp. *Culex tarsalis* *Psorophora ciliata* *Psorophora cyanescens*
Rio Grande River, Site 5 (Los Sauces)	July 5, 7	*Psorophora discolor*
Bluetown	July 5	*Psorophora confinnis*
Boca Chica, Site 1	July 5	*Aedes sollicitans* *Aedes thelcter* *Psorophora discolor*
	July 9	*Aedes sollicitans* *Aedes taeniorrhynchus* *Aedes thelcter* *Anopheles crucians* *Deinocerites pseudes* *Psorophora confinnis* *Psorophora discolor*
Laguna Vista, Site 6	July 6	*Aedes sollicitans* *Aedes taeniorrhynchus* *Psorophora discolor*
San Pedro, Site 31x	July 7	*Psorophora confinnis*
Valley View, Site 17	July 8	*Aedes sollicitans*
Boca Chica, Site 32x	July 9, 10	*Aedes sollicitans*
	July 11	*Aedes sollicitans* *Psorophora discolor*
Boca Chica, Site 33	July 9	*Psorophora discolor*
	July 11	*Aedes sollicitans* *Psorophora discolor*
Boca Chica, Site 33x	July 9	*Aedes sollicitans* *Aedes thelcter* *Psorophora discolor*
	July 10	*Aedes sollicitans* *Psorophora confinnis* *Psorophora discolor*
Boca Chica, Site 34x	July 9	*Aedes sollicitans* *Aedes taeniorrhynchus* *Aedes thelcter* *Psorophora discolor*
Brownsville, Site 33	July 9	*Aedes sollicitans*
Brownsville, Site 34	July 9	*Aedes thelcter*
Brownsville, Site 35	July 9	*Aedes sollicitans* *Psorophora confinnis*
Boca Chica, Site 40x	July 10	*Aedes sollicitans* *Psorophora discolor*
	July 11	*Aedes sollicitans*
Boca Chica, Site 41x	July 11	*Aedes sollicitans*
Boca Chica, Site 43	July 11	*Aedes sollicitans* *Psorophora discolor*
Corpus Christi, T1, 2, 3	July 15-19	*Aedes sollicitans*
Corpus Christi, T4, 6, 7	July 15-19	*Aedes taeniorrhynchus*
Corpus Christi, T5, 8	July 15-19	*Aedes sollicitans* *Psorophora confinnis*
Mission, Site 19	July 19	*Psorophora confinnis*
San Ygnacio[1]	July 22	*Anopheles pseudopunctipennis* *Aedes thelcter*
Boca Chica, (sites of wild mammal collections)	July 23	*Aedes sollicitans*
Devils' Lake Reservoir, Del Rio[2]	August 18	*Psorophora confinnis*

[1] San Ygnacio in Zapata County is located close to the Rio Grande River 205 miles northwest of Brownsville.

[2] Del Rio in Val Verde County is adjacent to the Rio Grande River, approximately 200 miles northwest of San Ygnacio, and 384 miles northwest of Brownsville.

The collections at Del Rio were made and identified by the Walter Reed Army Institute of Research. All other collections, identifications, and virus isolations were made by CDC, Atlanta.

1964. Although VEE virus was not isolated, it should be noted that mosquitoes collected at the same time and place yielded only one isolation in 1963 and none in 1964 *(33)*. Thus, it cannot fairly be concluded that *Culicoides* is not a vector. On the other hand, VEE virus reportedly was isolated from *Culicoides* during the epidemic in Ecuador in 1969.

Investigations during the summer of 1971 revealed that *Culicoides* (primarily *C. arubae)* and *Leptoconop* sp. were commonly found attacking horses in coasta areas of southern Texas. These species were found on infected premises and in areas where they were no appearing in the mosquito light traps *(20)*.

Investigators frequently have indicated that othe blood-feeding arthropods may be more than casua vectors of VEE virus. These often require differen methods for optimal surveillance and trapping. Mos work, however, has been limited to mosquitoes.

Morbidity and Mortality

Throughout the history of VEE in South Americ Central America, and Mexico at least four elements hav consistently limited accurate evaluations of the disease: difficult environment, poor surveillance, lack of trained observers, and exaggeration of losses. Variations of these elements during the epidemic in Texas similarly limited the validity of morbidity and mortality data. These variations included: nonspecificity of clinical signs, undetected subclinical disease, time lags from detection of clinical signs to receipt of laboratory confirmation, reporting rumors as fact, and the tendency to dramatize disaster by ascribing all sickness and death to the current popular disease.

Hidden Infection

It is evident that epidemic VEE was in the United States before its recognition. How long before is indeterminable.

The explosive effects of certain viruses such as African swine fever and rinderpest are notorious. These spectacular displays, it is now realized, are exceptional phenomena. The more prevalent manifestations, and those having the greater economic impact, are mild and insidious *(27)*. These latter viruses may appear as complexes involved in an interplay of environmental, biological, and climatological factors. Often they evoke few if any clinical signs in the host. The endemic forms of VEE virus may be of this nature. As long ago as the Trinidad outbreak of 1943 it was demonstrated that some equines can develop a natural immunity to VEE. A light initial attack by mosquitoes carrying VEE virus can produce subclinical infection followed by active immunity.

Until the detection of antibodies among indigenous Indian residents of south Florida, VEE in the United States was a hidden infection. Since the Indians were a permanent, relatively nonmobile population, infection had to be brought to them. Again the time sequence is speculative.

The records of the Texas outbreak reveal that some horses were noticeably sick at the time of vaccination. Predictably many became worse afterward. Many more that sickened or died had no visible signs of sickness when they were vaccinated. Horses frequently may develop high levels of viremia from VEE before signs appear. Initially in the task force program, failure to take this into consideration confused the assessment of VEE. Unusual signs following vaccination were minimized and classified separately from unvaccinated cases of suspected clinical VEE. This procedure arbitrarily dismissed the possibility of a horse incubating the true disease at the time of vaccination.

Another diagnostic practice minimized the significance of clinical signs other than those of the central nervous system. Respiratory signs, for example, widely prevalent preceding other common signs of VEE were dismissed as flu or strangles. If these signs were not accompanied by others suggestive of VEE, the investigation proceeded no farther. These respiratory manifestations, however, could have been part of the VEE syndrome. Among 851 unvaccinated horses with clinical signs suspicious of VEE, a total of 35 percent manifested either or both respiratory distress and nasal-ocular discharges. (Investigators in Trinidad and in Mexico also observed the appearance of respiratory difficulty and the development of a heave line as evidence of early brain damage associated with VEE.)

The importance of the omission is measured by the knowledge that VEE virus persists in the bloodstream well into the terminal stages of disease, the infected equine remaining a continuous source of potential infection; 50 percent of VEE cases may show no clinical signs whatever; and clinical VEE does not always manifest central nervous system involvement *(22, 30, 26)*.

Reporting

Hundreds of thousands of equine deaths with mortality as high as 80-90 percent were reported during the VEE epidemics that swept South and Central America and Mexico. In the absence of reasonably valid reporting systems these estimates are suspect, if not as raw data, certainly as reliable mortality rates attributable to VEE. Too many diseases share signs similar to those of VEE to permit deducing death rates from signs alone. And in many countries where nutritional levels are low and management is indifferent, spectacular disease is not the principal cause of death, though it may often be indicted for it. In those circumstances conceivably millions of equines live in a continuing state of malnutrition, parasitism, and subhealth which under stress may produce death. (According to horse owners many animals that sickened or died during the epidemic in Texas were malnourished due to prolonged drought and poor range.)

In referring to the Mexican epidemic of 1970-71, the Director of the Faculty of Veterinary Medicine at the National University of Mexico said that reporting by Mexican newspapers had been "exaggerated and alarmist." The official estimates of equine deaths from VEE in Mexico were 15,000-20,000 in a total equine population of approximately 9 million.

Reporting of VEE in the United States was similarly afflicted with exaggeration and inaccuracy. On July 14, a spokesman for the task force in Harlingen estimated a total of 500 cases of sick, recovered, or dead horses, a figure in itself incapable of verification. A national news service immediately converted this estimate into 500 "dead" horses, and the error became the accepted base figure for subsequent misquotation. Twenty-four hours later the official figures had become "800 dead horses and more than 1,000 with the disease."

It was essential to develop a disease intelligence network. It was impossible to see every sick animal or to confirm each death by laboratory test. Hence, horse owners and handlers were requested to report cases of recently sick horses to a veterinarian, county agricultural agent, or State or Federal animal health official. Thus, out of practicality, reported figures of all equine sickness and death became the official estimates for VEE. While acceptable for the mass media, this practice actually diminished the validity of conclusions about the true disease. According to official estimates, 1,528 horses died from all causes during the VEE epidemic. Conceivably, not all cases of epidemic VEE were seen or confirmed, despite the fact that 1,563 cases were investigated. In 9 percent of the investigations samples either were not taken, were broken, spoiled, or lost in transit, or were still awaiting final laboratory tests (as of December 31, 1971). Within these limitations, 142 equine deaths from epidemic VEE were confirmed in 75 herds in 26 Texas counties (table 7 and fig. 6).

Clinical Signs

Another factor that clouds estimates of morbidity and mortality for VEE not substantiated by laboratory confirmation is the variability of clinical signs. Variations in signs may be due to geography; previous exposure to the disease; age; the presence of secondary

Table 7.—Summary of epidemic VEE in horses—Texas, June 29-November 11, 1971

County	Herds	Total in these herds	Horses with Encephalitis	Horse deaths	Date specimen collected	
					First positive case in county	Last positive case in county
Live Oak	1	26	1	[1] 1	6-29-71[1]	6-29-71
Cameron	13	52	13	6	7-1-71[2]	7-14-71
Hidalgo	11	113	35	12	7-7-71	7-13-71
Nueces	3	52	7	1	7-10-71	7-13-71
Aransas	8	26	8	1	7-11-71	7-15-71
San Patricio	4	25	5	1	7-11-71	7-13-71
Calhoun	2	3	0	2	7-12-71	7-18-71
Willacy	1	4	0	1	7-19-71	7-19-71
Refugio	3	132	4	1	7-14-71	7-20-71
Harris	2	39	1	1	7-15-71	7-21-71
Brooks	1	92	7	6	7-16-71	7-16-71
Victoria	1	40	1	7	7-19-71	7-19-71
Guadalupe	1	40	2	1	7-19-71	7-19-71
Val Verde	1	33	10	6	7-25-71	7-25-71
Brazoria	2	16	1	1	7-27-71	8-12-71
Matagorda	1	24	1	0	7-30-71	7-30-71
Maverick	3	138	16	68	8-6-71	8-31-71
Dimmit	4	49	8	4	7-23-71	8-31-71
Zapata	4	404	5	13	8-4-71	9-2-71
Edwards	2	11	2	0	8-28-71	8-28-71
Webb	1	2	1	0	8-26-71	8-26-71
Zavala	1	5	0	3	9-8-71	9-8-71
Crockett	2	28	7	4	9-14-71	10-4-71
Jim Hogg	1	150	2	1	7-14-71	7-14-71
Presidio	1	4	1	1	9-11-71	9-11-71
Starr	1	1	1	0	11-7-71	11-7-71
TOTALS	75	1,508	139	142		

[1] First clinical signs seen 6-24-71.

[2] First clinical signs seen 6-15-71.

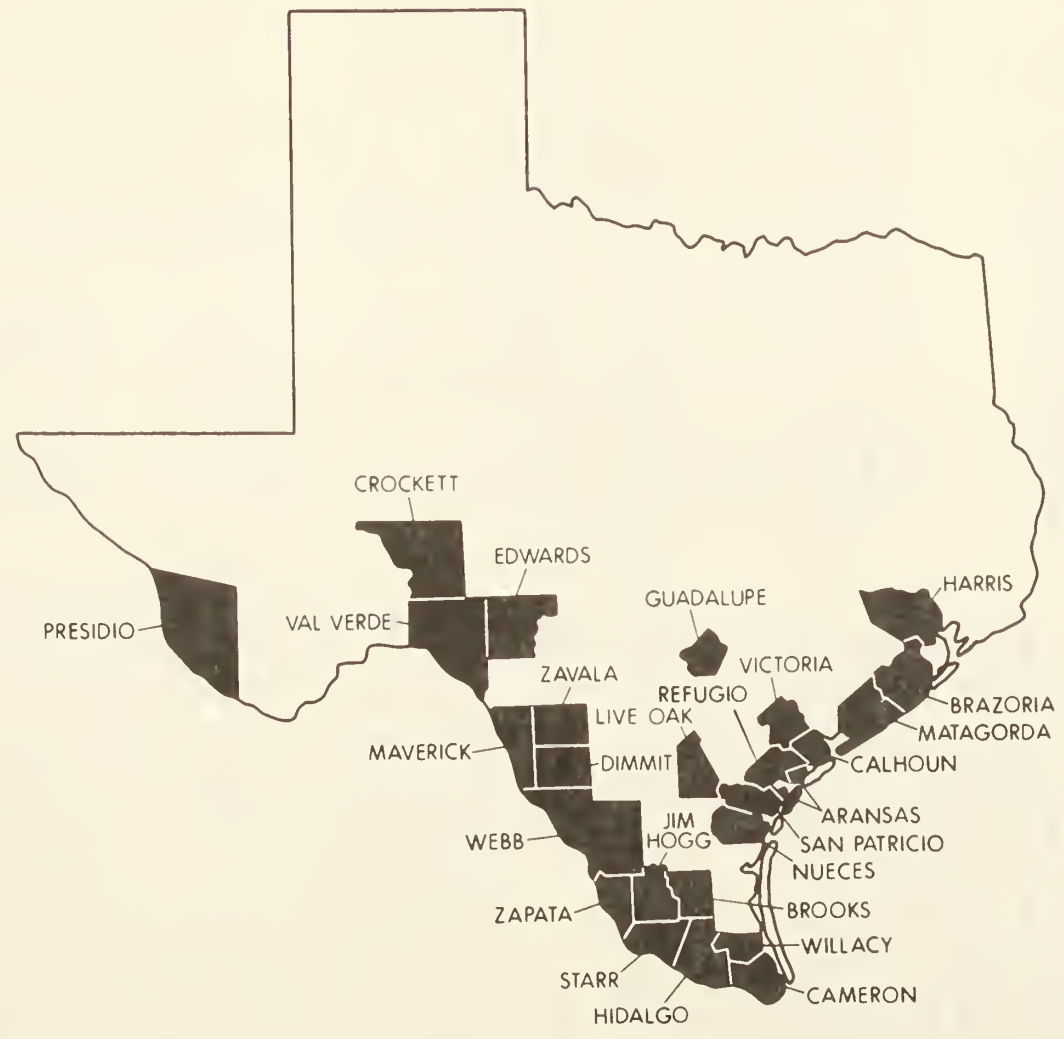

Figure 6.—Counties in which one or more isolations of epidemic VEE virus were made June 29 - November 11, 1971.

bacterial invaders, of enteroviruses or arboviruses other than VEE; concommittant conditions of malnutrition and subhealth; or whether the clinical signs resulted from natural infection or laboratory experiment. The fact, previously mentioned, that about 50 percent of equines positive for VEE show no signs, is another point.

Trinidad.—Under experimental conditions using naturally-infected mosquitoes, the incubation period of the virus was 48 hours. Death of donkey hosts occurred in 6 days.

The signs for the first 48 hours were inappetence, depression, and a temperature of 103°-105° F. Thereafter, an early sign was respiratory difficulty (as evidenced by reflex abdominal breathing) and neurological signs (somnolence, excitability, and incoordination or drifting gait). The animal became prostrate, continually simulated "trotting" movements, and had chewing spasms.

Sometimes the animal rose and plunged violently. It might circle. Swallowing was difficult. The animal stood with head (and penis) hanging, depressed. However, this rarely progressed to somnolence and the patient responded to physical stimuli *(16)*.

Mexico.—Under natural conditions the incubation period is from 1 to 3 weeks. Viremia, apparent 22 hours after inoculation of virus. may persist until death. Virus is present in the saliva, nasal and ocular secretions, and in the milk.

The first significant sign may be hypersensitivity to touch and sound. There may be excitement, apparent blindness, and difficulty in swallowing. The development of a heave line may be the most prominent sign. (This results from contraction of the greater oblique muscle, evidence of damage to the respiratory center of the brain.)

The horse walks in circles (sometimes in a figure-8 pattern) stumbling over and against objects however small. There are involuntary muscle spasms of the shoulders and face, and penile erection.

The horse is depressed and stands with its head down as though asleep. Mouthfuls of half-masticated food are held between the lips. There is pupillar reflex to light.

The horse is unable to lift its head, pressing it against objects. It supports itself by crossing the legs, pressing against a solid object, or pulling on a halter rope. There are chewing movements, paralysis of the pharynx, and blindness. Urination and defecation are suppressed.

General paralysis indicates the terminal phase. The horse falls and cannot rise. Death usually comes 2 to 4 days after appearance of the first signs.

Some horses do not become paralyzed and survive. But they may be affected mentally and do not adequately respond to stimuli *(30)*.

During the epidemic that originated in the Municipio of Tamiahua, Veracruz, in April 1971, the most frequent clinical signs were: inappetence, elevated temperature, nasal exudation, depression, rigid neck muscles, extreme weakness, circling, and visual impairment. Clinical histories frequently were confused by the presence of heavy parasitism and anemia.

United States.—Ten of the most common clinical signs seen in three categories of horses in southern Texas are compared in table 8. The first group consists of vaccinated and unvaccinated horses positive for epidemic VEE. The second group comprises unvaccinated horses with clinical signs suspicious of VEE. The third group consists of vaccinated suspects.

Some signs were observed by diagnosticians and epidemiologists, others were reported by owners or caretakers. Since the horses were not under constant surveillance some signs undoubtedly were missed. On occasions, a recumbent, dying horse was the first and only sign seen.

The three most common signs seen in all groups of horses except one were depression (19-53 percent), inappetence (11-41 percent), and high temperature (11-35 percent). Depression included the gamut of signs from sleepiness to stupor.

Among nonvaccinates in herds positive for VEE, impaired vision took the place of inappetence. This sign was also among the three most commonly seen among horses with VEE in Mexico.

The next group of signs seen were incoordination (9-28 percent), circling (6-33 percent), chewing movements (6-30 percent). Among nonvaccinated suspects, 21 percent were immobile or reluctant to move and 20 percent had a braced stance. Hypersensibility to touch and sound, considered to be the most significant sign reported in horses affected with VEE in Mexico, either was not seen or was overlooked by observers in Texas.

Circling is commonly believed to be an important clinical sign in the detection of VEE. But even equines with epidemic VEE do not all circle. As has been previously noted, the mare that became the first case recognized in Texas did not circle. Among the three groups compared in table 8, circling was seen in 6 to 17 percent of nonvaccinated horses, but among 6 to 33 percent of the vaccinated horses. The unreliability of this sign in the clinical picture of VEE is further evident from the fact that circling frequently accompanies colic from eating mesquite beans, a phenomenon seen annually in Texas.

When clinical signs of VEE appeared in vaccinated horses, from 40-87 percent in all groups recovered

Table 8.–Comparison of 10 most common clinical signs seen in three groups of horses, South Texas, June 23-August 19, 1971

Vaccinates and nonvaccinates in herds positive for epidemic VEE				*Unvaccinated suspects*		*Vaccinated suspects*	
Total horse population	654			851		3,284	
Sick	30			75		133	
Dead	57			84		199	
Vaccinates (453)		*Nonvaccinates (201)*					
Clinical sign	*Percent*	*Clinical sign*	*Percent*	*Clinical signs*	*Percent*	*Clinical signs*	*Percent*
Depression-stupor	19	Depression-stupor	22	Depression	53	Off-feed	41
Off-feed	11	High temperature (106.6° F.)	18	Off feed	41	Depression	40
High temperature (107.2° F.)	11	Impaired vision	11	High temperature	35	High temperature	33
Recumbency	10	Incoordination	10	Incoordination	27	Circling	33
Impaired vision	10	Braced stance	9	Chewing movements	24	Chewing movements	30
Difficult breathing	10	Off feed	8	Immobile-reluctant to move	21	Incoordination	28
Incoordination	9	Chewing movements	7	Braced stance	20	Impaired vision	26
Circling	6	Circling	6	Difficult breathing	18	Restless-excitable	22
Chewing movements	6	Restless-excitable	4	Circling	17	Impaired	20
Nasal/ocular discharge	6	Difficulty in breathing	4	Nasal/ocular discharge	17	Drooling	20

within a matter of hours up to 5 days. When clinical signs among nonvaccinates ended fatally, 75-92 percent of the deaths occurred within the same period of hours up to 5 days. (table 9).

A separate analysis was made of the group comprising 654 vaccinated and nonvaccinated horses in 32 nonrandomized herds positive for VEE. These data are summarized in table 10 including parts A, B, C, and D.

Table 9.–Comparison of time interval from appearance of first clinical signs to recovery or death in three groups of horses, South Texas, June 23-August 19, 1971

	Epidemic VEE in herd				*Unvaccinated suspects*		*Vaccinated suspects*	
Total horse population	654				851		3,284	
Time (days)	*Horses recovered Percent*		*Horses dead Percent*		*Horses recovered Percent*	*Horses dead Percent*	*Horses recovered Percent*	*Horses dead Percent*
	Vaccinated	*Non-vaccinated*	*Vaccinated*	*Non-vaccinated*				
<1-5	40	-	76	75	71	92	87	80
6-10	60	-	24	17	7	8	12	20
14-18	-	-	-	-	7	-	1	
23-30	-	100	-	-	-		-	
32-38	-	-	-	-	7		-	<1
No information				8	8			

Table 10.–Summaries of data concerning a total of 654 vaccinated and nonvaccinated horses in 32 nonrandomized herds positive for epidemic VEE, South Texas–June 23-August 19, 1971

A. Days from Clinical Signs to Recovery or Death

	Vaccinates		*Nonvaccinates*	
	Days	*Percent of horses*	*Days*	*Percent of horses*
Recovery	3-7	100	23-30	100
Death	<1-3	68	<1-2	50
Death	5-7	32	4-8	50

Among the vaccinated horses in these herds with epidemic VEE, all horses showing clinical signs recovered within 3-7 days. For the nonvaccinates affected in those same herds recovery took between 23 and 30 days.

Deaths in both vaccinates and nonvaccinates occurred within two distinct time periods. From 50-68 percent of the deaths occurred within a matter of hours up to 3 days. The second group of deaths, involving from one-third to one-half of both groups of horses, occurred between 4 and 8 days after the appearance of the first clinical signs.

B. Days from Vaccination to First Clinical Signs

Days	*Percent of horses*	
<1	10	52 percent
1	0	
2	31	
3	10	
4	0	
5	0	
6	0	
7	3	10 percent
8	7	
9	0	
10	10	
12	3	
33	3	22 percent
40	3	
41	3	
42	3	
46	3	
48	7	

Clinical signs of VEE appeared in less than 1 day to 3 days in 52 percent of the vaccinated cases. Ten percent appeared between 7 and 8 days. A third group of horses, representing 22 percent of the population, showed the first clinical signs of VEE between 33 and 48 days after vaccination. (The latter represent a single ranch of approximately 400 horses with a long history of "strangles". The ranch also contains 675 cattle, buffalo, giraffes, zebras, ostriches, 20 varieties of deer, and a cheetah. At least 10 horses and one 16-week old giraffe died. Half of the dead horses were under 7 months of age, four were between 1 1/2 and 2 years old. Deaths were sudden. Vaccination was conducted on two separate occasions, and presumably some of the animals were incubating the disease at the time. All horses were depressed following vaccination and within a day lost 25-35 pounds. The history of "strangles" may have included undifferentiated VEE with the development of partial immunity in the herd. The presence of a large number of other species as potential hosts may also be associated with the delayed appearance of clinical signs as well as the large number of deaths in the herd. Improperly administered vaccine could have given incomplete immunization.)

C. Age and Sex of Sick or Dead Horses

Age in Years	*Vaccinates*		*Nonvaccinates*	
	Sick	*Dead*	*Sick*	*Dead*
	Percent		*Percent*	
<1	25	22	0	36
1-3	13	44	36	36
4-7	13	6	27	0
8-11	36	16	9	14
12-15	13	6	18	7
16-20	0	6	9	0
20>	0	0	0	7
Total Males	14	38	50	64
Total Females	86	62	50	36

The age group under 1 year accounted for 22-36 percent of the deaths in both vaccinates and nonvaccinates. Sickness in that same age group was limited to 25 percent of the vaccinates only. Though 36 percent of the nonvaccinates under 1 year of age died, death apparently was so sudden that signs of illness were not observed. Among nonvaccinated horses 36 percent of those between 1 and 3 years of age sickened and an equal percentage died. Among vaccinates in the same age group, on the other hand, more than 3 times as many died as were sick. The latter would seem to indicate that the horses were sick either with VEE or some other disease before they were vaccinated.

The decline in mortality in the age group from 16 years and beyond (6 percent among vaccinated horses and 7 percent among nonvaccinates) is expected. Even if the incidence was the same at all ages, fewer horses are alive in the later years.

Among male horses, both vaccinated and nonvaccinated, more died than were noted sick. On the other hand, while the number of sick female horses was either equal to or considerably greater than the number of sick males, more females than males recovered.

D. Morbidity and Mortality

Total herds: 32
Total horse population: 654

	Population at risk	*Sick (recovered)*	*Dead*	*Morbidity rate*	*Mortality rate*	*Case fatality rate*[1]
				Percent	*Percent*	
Vaccinates	453	5	22	6	5	81
Nonvaccinates	201	2	12	7	6	86

---

[1] Probability that a sick horse will die.

Recovery After Treatment

The exaggerations since 1935 of sickness and death from VEE have created the impression of an implacable killer. While it is undeniable that considerable losses have accompanied VEE, the records tend to discredit the specific indictment of VEE.

The value of symptomatic treatment and improvised nursing case in encephalitis was experimentally demonstrated among 75 mules in Trinidad in 1945. The mules were confined in covered sheds with bedding. Troughs for feed and water (with added molasses) were elevated within easy reach. Chloral hydrate (2 ounces) was administered via stomach tube to control violence. Citrated convalescent whole blood was administered intravenously. Although the mortality from the disease in Venezuela was 83.29 percent, mortality in the experimental group was only 25 percent.

During the VEE epidemic of 1946 in Ecuador morbidity and mortality were not high. Frequent cases of improvement were reported following palliative treatments such as purges, bloodletting, and injection of intravenous urotropine *(35)*. Although VEE virus was isolated from a sick horse, it is not certain that all cases of sickness with recovery following these treatments were VEE. This, of course, is the converse of attributing all sickness and death to VEE.

Symptomatic treatment by owners and veterinarians in southern Texas undoubtedly reduced losses. Such simple procedures as keeping horses on their feet, spraying with a hose to prevent dehydration, and assistance in eating and drinking frequently were associated with recoveries. Veterinarians administered massive doses of steroids. Vitamin B complexes, glucose, and antibiotics.

The rational for treatment was that the multiplication of the VEE virus paved the way for entry of bacterial agents which produced certain clinical signs and magnified others. Administration of antibiotics and other supportive treatment controlled the secondary bacterial agents which in some cases seemed to be sufficient to permit recovery from the viral infection.

From observation and hearsay many horses that died were malnourished from drought-stricken pastures and neglected. In some cases mortality seemed to be aggravated by the indifference of owners. In the marshy coastal areas, for example, deaths ascribed to VEE occurred when recumbent horses were left to drown.

Movement of Horses, Humans, and Vehicles

The Rio Grande River is a tenuous national boundary. It has never been an economic, cultural, or social barrier. Day and night there is constant movement of humans, animals, and vehicles in both directions across the border. Cattle and horses frequently wander across the river of their own volition. Occasionally the movement of livestock is clandestine. Most of these are apprehended, a few are not.

From experiences with VEE in South and Central America, it was concluded that the movement of viremic but asymptomatic horses to noninfected areas was the most common means of dissemination of the disease. While strict control of the movement of horses is sought, it is not always achievable. Conceivably, VEE in 1971 was spread by the movement of horses within Mexico, as well as from the Lower Rio Grande Valley into other parts of Texas.

The first confirmation of epidemic VEE in 1971 (April) was made from the blood of a sick horse at Tampico Alto, Mexico. This horse was part of a shipment of six imported from northern Tamaulipas 3 months previously. This movement presents two hypothetical possibilities: the introduction of latent infection from the north, or the arrival in Tampico of a new susceptible population to rekindle existing infection. On its face, an interval of about 3 months would seem too long for the incubation of latent epidemic VEE brought from the northern part of the state. In past experiences endemic VEE was largely limited to wet lowlands and coastal areas. Explosive epidemics occurred in dry regions. However, there is considerable uncertainty about the mechanism of incubation and transmission in areas of concurrent endemic and epidemic infection among populations of equines only partially immunized.

Concerning the movement of horses across the Rio Grande River from Mexico, the U.S. Customs Service reported eight separate apprehensions involving 13 strayed or smuggled horses and one legal entry of two horses between March 16 and July 11 (table 11). These incidents covered an area from Brownsville 20 miles north to San Benito, and 125 miles northwest along the Rio Grande River. Neighbors were the source of a report of two clandestine ("wet") horses brought from Mexico in June 1971 to a stable near Brownsville where they died. Two horses were found positive for equine piroplasmosis (no other tests were run) and were destroyed by animal health authorities.

Table 11.—Movement of horses across the Rio Grande River, March 16-July 11, 1971

Date	Location where found in Texas	Number	Origin	Nature of movement	Disposition
March 16	Donna	1	Mexico	Stray	Sold
March 17	Brownsville	1	Mexico	- - - - - do - - - - -	Died—cause unknown
April 14	25 mi. W. Rio Grande City	5	U.S. to Mexico to U.S.	- - - - - do - - - - -	Released to owner
May 5	20 mi. S.W. Rio Grande City	2	Mexico	- - - - - do - - - - -	Claimed by owner
May 20	San Juan	1	U.S. to Mexico to U.S.	- - - - - do - - - - -	Claimed by owner and died—cause unknown
June 21[1].	San Benito	1	Mexico	- - - - - do - - - - -	Piroplasmosis positive—destroyed
July 7	San Benito	1	Mexico	- - - - - do - - - - -	Piroplasmosis positive—destroyed
July 11.	Brownsville	1	Mexico	- - - - - do - - - - -	Unknown
June 18[2].	Brownsville	2	Mexico	Imported	Placed on ranch with 8 native horses

[1] At this time inspectors of the Mounted Tick Force patrolling the river reported seeing horses circling on the Mexican side, opposite a point 1 mile east of Hidalgo, Tex.

[2] Border officially closed this date to entry of horses from Mexico. Eight horses in this shipment were refused entry for health reasons. On July 17, one of the native horses (vaccinated on June 30) also was coughing and had a profuse nasal discharge. A second horse on this ranch (vaccinated on June 30) had respiratory distress, nuchal rigidity, muscle spasms, and was circling. Diagnoses were not confirmed.

In mid-June, following unconfirmed reports of VEE in villages 16 miles south of Matamoros, and before the vaccine was released in Texas, State authorities confirmed that horses were being moved out of the Valley in truckloads to San Antonio, Ft. Worth, and Victoria. An extreme example was the reported shipment of a valuable Palomino mare as far as Florida. Since VEE was not recognized outside Texas, it is presumed the early, long distance movement of horses did not spread the disease, at least at that time. The local movement of exposed horses is more suspect. The danger of transmission of VEE arises from two possibilities: the introduction of a viremic horse among susceptibles upon arrival at its destination and the continuous exposure of the horse to blood-consuming arthropods enroute.

A vaccinated horse was moved on June 29 from Brownsville to Graham in northcentral Texas. The horse died on July 4 without a confirmed diagnosis. An unvaccinated horse was transported on June 21 from a ranch near Beeville to Corpus Christi. The horse died with a confirmed diagnosis of epidemic VEE. It is not possible either to establish all the movements of horses within southern Texas or to document those which were instrumental in transmitting infection. Some horses move to breeding farms for several months at a time. Approximately 10 percent of the preponderant pleasure horses in the populated areas attend weekly horse shows and rodeos. This may involve travel of up to several hundred miles.

One of the more popular youth rodeos in southeastern Texas was the Saturday night Rio Round-Up at Edinburg. Approximately 70 horse owners regularly participated. The overwhelming majority had their horses vaccinated in June. In compliance with the quarantine on the movement of vaccinated horses, the Rio Round-Up was closed from July 3-24. Thereafter vaccination certificates were required for admission. Several horses were reported sick after vaccination, but all recovered.

F.S. was one of the few Round-Up participants who had not vaccinated his horses. On July 4, he noticed that his unvaccinated 3-year-old mare was sick. That same day F.S. had the mare's colt vaccinated. The colt died on July 9. The mare died on July 10 and epidemic VEE virus was subsequently isolated from her brain.

The F.S. ranch was located in an irrigated area south of Alamo, near the Rio Grande River, where approximately 50 horse deaths were reported. Infection probably was acquired at the home premises.

This incident does not minimize the importance of the movement of viremic horses in the spread of VEE. It rather suggests that this particular rodeo, despite the opportunity for contact exposure, was not a serious focus for the spread of infection—either because vectors were not available or vaccination protected the susceptible population.

Human agents and vehicles may serve as conveyers of infected vectors. Another possibility is the transmission of live virus on fomites such as shoes, clothing, baggage, tires, and in the contents of commercial vehicles. Some residents of southern Texas own ranches or businesses in Mexico which they visit daily. Others make frequent visits to relatives in rural areas. In June and July 1971, many individuals from Texas visited places in northern Mexico where mosquitoes and sick and dead horses were seen in great numbers. Subsequently sick and dead horses appeared at or near some of the respective premises in Texas. The evidence is circumstantial and difficult to evaluate; nevertheless, it is part of the disease pattern. Two examples will make the point.

L.C. owns a ranch near San Benito. In early June for 5 days he and his wife visited San Carlos, a village located 1 1/2 hours by vehicle south of Ciudad Victoria. (L.C. and his wife visited San Carlos again on July 24, returning to Texas on July 28 with reports of sick humans and many sick and dead horses lying in the fields.)

In the afternoon of July 6, L.C. noted his 25-year-old male horse making chewing movements and trembling. No other signs were seen. Next morning the horse was found in the canal adjacent to the house. It was pulled out with a wrecker but died immediately afterward. Epidemic VEE was ultimately isolated from the brain.

The second illustration concerns B.C., who owns a ranch also located near San Benito. On July 10, B.C. vaccinated 15 horses. One 14-year old female horse that was noted standing braced, immobile, and twitching was not vaccinated. The unvaccinated female died that evening and was buried by the county authorities. No samples were obtained and no confirmation of the disease was made. On July 14, the first of six vaccinated horses became sick. All six ultimately recovered within 5-6 days. (B.C. had had aching muscles and headache during the week the horses were sick.) B.C. (who had had several years of college training directed toward the study of medicine) operated a part-time enterprise selling and distributing blocks of mineral and salt mix. During epidemiological investigation B.C. stated that he probably was responsible for spreading VEE. His involvement was related to his part-time business. B.C. made deliveries as far as Beeville (see the First Case, page 9). On the return trip he filled his truck with feed collected at San Antonio for delivery to local mills in San Benito. More important, B.C. customarily made three or four trips a week throughout the Brownsville area and its environs. This practice continued during the epidemic. Deliveries were made in a closed truck to a number of premises on which horses had sickened or died. Among these were several trips to the D.P. premises, on which the first case of epidemic VEE in the United States was diagnosed. The last trip to those premises had been made about one week before sickness appeared there.

Disposal of Dead Horses

By the second week in July the increasing death toll was visible in the pastures of southern Texas and in the Rio Grande River. Statute obligated owners to dispose of carcasses by deep burial under 5 feet of earth with prior slashing and liming of the carcass, or disposal in a sanitary land fill. Incineration was not permitted.

County commissioners, sanitarians, and public utilities offered sites and heavy equipment to help in the disposal of carcasses. (The road superintendent, working under the County Commissioner of Cameron County, buried 41 horses during July and August.) But the early accumulation of dead equines inevitably caused delays. Unburied carcasses lying in the fields for days provided potential foci for the spread of virus by arthropod vectors, dogs, rats, and carrion-eating mammals and birds. Many owners did not wait for the official disposal crews but breached the statute and burned their dead equines. This provided another means for the possible dissemination of VEE. Virus could have been released as aerosols to be spread via air currents and winds. Experimental evidence indicates that VEE virus particles of 1 to 5 mμ are highly stable in aerosol form. The virus may persist for extended periods of time and under proper conditions could become a source of airborne infection *(13)*.

Wildlife

The determination of the whereabouts of VEE virus, and the manner in which it is maintained in nature during inter-epidemic periods, has confounded investigators for 30 years.

The first attempts to find a reservoir for the virus in nature were made in Trinidad in 1943. A wild deer, a captive monkey, chickens, buzzards, and vampire bats were the subjects. Nothing definitive was determined *(16)*.

Birds

The association of VEE with water courses and swamps suggested an ecological relationship with birds: migratory birds for the long-distance dispersion of virus and resident or nesting birds for local spread.

Neutralizing antibodies to VEE virus were demonstrated in the sera of eight species of birds caught in the swamplands of Louisiana in 1955 (table 12). Two of these, the white ibis and the yellow-crowned night heron, migrate from South and Central America in the summer.

Subsequently VEE virus was isolated from nine species of birds caught in northwestern Panama. Several of these birds migrate to North America.

Table 12.–Wildlife associated with Venezuelan Equine Encephalomylitis South Texas, June-November 1971

Host	*Species*	*Location*	*Date collected*	*Collector*	*Test*[1]	*Laboratory*
Black-tailed jackrabbit	*Lepus californicus*	Cameron County	July and August	CDC, Ft. Collins	HI, NT	CDC-Ft. Collins
Cactus wren	*Camplorhynchus brunneicapillus*	Boca Chica	--- do ---	CDC, Atlanta	NT	CDC, Atlanta
Cotton-tail rabbit	*Sylvilagus* sp.	Boca Chica	--- do ---	-------- do --------	NT	Do.
Desert cotton-tail	*Sylvilagus audubonii*	Cameron County	----do ---	CDC, Ft. Collins	HI, NT	CDC-Ft. Collins
Gray fox	*Urocyon cineroargenteus*	Graham Ranch, Corpus Christi	July 23	-------- do --------	Epidemic VEE virus	CDC, Atlanta
Opossum	*Didelphis* sp.	Boca Chica	July and August	CDC, Atlanta	NT	Do.
Opossum	*Didelphis* sp.	Stell-Lind Banco, Boca Chica	July 20	-------- do --------	Epidemic VEE virus	Do.
Pack rat	*Neotoma* sp.	Boca Chica	July and August	-------- do --------	NT	Do.
Cabot's (sandwich) tern	*Thalasseus sandvicensis*	Flour Bluff, south of Corpus Christi	August 8	Sch. of Pub. Health Univ. of Texas	Epidemic VEE virus	Do.

[1] HI = hemagglutination inhibition,
NT= neutralization.

Further studies were conducted during the epidemic in Venezuela of 1962-64. Although VEE virus was isolated from two domestic chickens, it was not found among wild fowl in the same endemic area. Chicks under 1 month of age died from experimental infection. Those over 1 month developed antibodies with no visible illness. (It will be recalled that sick chickens were seen in El Moquete, 16 miles south of the United States border in an area of equine illness and death, and human sickness, late in June 1971. Sick chickens and dead wild birds were also reported on the D.P. premises, site of the first case confirmed in the United States. In neither instance were antibody determinations or virus isolations attempted.)

The implication of birds in the transmission of VEE led to investigation of the role of mosquitoes in the infection cycle. Experimental tests were conducted with cardinals, three species of sparrows, domestic pigeons, and *Aedes triseriatus* mosquitoes *(4)*.

Blood virus titres in birds were considerably lower than in mammals and infectious levels for *Aedes triseriatus* mosquitoes persisted for only 1 or 2 days compared with up to 4 days in horses. The studies showed, nevertheless, that birds can be infected by mosquitoes and show no signs of disease. Conversely, mosquitoes may acquire infection by feeding upon infected birds.

Pigeons were reported susceptible to respiratory infection with VEE virus, posing the possibility of virus transmission from bird to bird by means of aerosols.

Thus, though their role is still not clearly defined, birds may be involved both in the maintenance of endemic infection and as foci for epidemic spread.

Since 1946, at one time or another, 345 bird species have been recorded in the Rio Grande Valley or on the south Texas coast. Some of these visitors are rare. Others, like the flycatchers, warblers, and vireos are transients. Three species, because of their migratory patterns and breeding habits, satisfy the qualifications of potential disseminators of VEE. White-wing doves constitute the largest migration from Central America. They form huge breeding and nesting colonies of up to 6 million birds in the native brushland of northeastern Mexico. More than 400,000 nest in the brush and citrus groves of the lower valley in Texas.

Several hundred thousand ducks and geese winter in the Valley and along the coast or tarry for a short period during migration. By the last of April most have departed. Two exceptions are the black-bellied tree duck and the fulvous tree duck.

The black-bellied tree duck winters in the Gulf Coast marsh areas of Mexico. Migrating north in April, about 4,000 nest until December in the Rio Grande delta upriver to Rio Grande City. Others nest north along the Gulf Coast to Corpus Christi. Their habitat is the ponds, resacas, marshes, and swamps. As its name implies, the duck frequently sits in trees. It nests in tree cavities and in swamps.

The fulvous tree duck winters in Central and South America. Approximately 20,000 pass the months from April to October in the marshes, ricefields, and ponds of the Texas coast. The species nests in grass-lined saucers in grass or marsh vegetations.

From July 19 to September 24, 1971, a total of 1,621 birds were collected at sites in Cameron County were VEE virus had been isolated in mosquitoes. VEE virus was not isolated from any of the birds collected. White-wings, black-bellied tree ducks, and fulvous tree ducks were not included in the collection.

VEE virus was isolated, however, from one species of bird, a sandwich tern collected on August 8, south of Flour Bluff, Corpus Christi. The collection was made by investigators from the School of Public Health, University of Texas at Houston. The virus was isolated and identified at CDC, Atlanta. (At the same time 863 mosquitoes primarily *Aedes sollicitans*, *Aedes taeniorrhynchus*, and *Psorophora confinnis* collected at Flour Bluff were negative for VEE virus.) Antibodies to VEE were detected in a cactus wren collected in the Boca Chica flats east of Brownsville by CDC investigators.

The sandwich tern, or Cabot's tern, is a gregarious bird. The race is widely dispersed in both the Eastern and Western Hemispheres. In the Eastern Hemisphere, the sandwich tern ranges from Great Britain to south of the Tropic of Capricorn. In the Western Hemisphere, the sandwich tern winters from Florida and the Bahamas, to the Caribbean and Atlantic coasts of South America between Colombia and Brazil. The species breeds from the southern Atlantic and gulf coasts of the United States to the Bahamas, West Indies, and the coast of British Honduras. A bird of this race provided one of the few records concerning the longevity of sea birds. Banded in Cumberland, England, in June 1917, the sandwich tern was recovered in southwestern Africa nearly 14 years later *(28)*.

The detection of VEE virus in one sandwich tern among almost 1,700 miscellaneous birds sampled is significant from the standpoint of knowing that an infected arthropod had fed on the bird. It gives no indication whether the tern is an important host in the cycle of maintenance and dissemination of the virus. The species breeds from April to July. The virus was collected on August 8. Since viremia in birds lasts only 1 to 2 days, the virus was undoubtedly acquired in Texas from the bite of an infected mosquito on the Gulf Coast.

If migratory birds have any significance in the cycle of VEE—which is still to be proven or disproven—failure to sample white-wing doves, black-bellied tree ducks, and fulvous tree ducks was unfortunate. First, because they winter in the marshy habitats in which VEE virus seems to arise and flourish, and second, because they migrate to breed and to nest in similar habitats in northeastern Mexico and southern Texas.

Mammals

The involvement of wild mammals as hosts of VEE virus has been studied since 1943. Several weeks before the outbreak in Trinidad, a wild deer exhibiting signs of paralysis and stupor, died. Encephalitis was diagnosed from examination of the brain, but no attempt was made at that time to determine the causative agent. Since then, investigations conducted in South and Central America, Mexico, Florida, Utah, and Texas have provided evidence of VEE virus or antibodies in approximately 72 species of wildlife (table 13).

Thirty-one species of wild mammals have been found in the Rio Grande Valley and in the coastal prairie of southern Texas. These include mountain lion, bobcat, ocelot, jaguarundi cat, white-tailed deer, fox, peccary, coyote, opposum, beaver, raccoon, armadillo, long-tailed weasel, striped skunk, and feral pigs; also four species of rat, five species of mouse, two species of rabbit, and one species of squirrel.

Exotic animals introduced but now running wild include nilgai, black-buck, aoudad, and fallow deer.

In the fall of 1970, there was an estimated total of 135,235 deer in Cameron, Hidalgo, Starr, Willacy, and Kenedy Counties. Hidalgo County has the largest overall wildlife population.

During the period July 19-September 23, 1971, a total of 1,703 mammals (and 271 reptiles) were collected in southern Texas. Epidemic VEE virus was isolated from two species, an opossum and a gray fox. The opossum an adult male, *Didelphis marsupialis*, was collected on July 20 at Stell-Lind Banco, east of Brownsville, by a survey team from CDC, Atlanta. The gray fox, *Urocyon cenereoargenteus*, was collected on July 23 at the Graham Ranch, Corpus Christi, by a team from the School of Public Health, University of Texas.

The opossum belongs to a terrestrial species that is found from southern Canada through the Americas to northern Argentina. It usually inhabits forested or brushy areas although it has been found in open country near wooded watercourses. The opossum passes the day

Table 13.—Animals associated with Venezuelan Equine Encephalomyelitis, 1938-1972[1]

Host	Species	Location	Year	Test
Mammals:				
Bat				
Big brown	*Eptesicus fuscus*	Ft. Detrick, Md.	1956	Laboratory Exper. (LE)
Big fruit-eating	*Artibeus literatus*	Panama	1959-62	Antibodies (Ab)
"Frugivorous"		Columbia	1967	Virus isolation
Little brown	*Myotis lucifugus*	Ft. Detrick, Md.	1956	LE
Long-eared	*Pipistrellus subflavus*	----do----	1956	LE
Long-tongued	*Glossophaga* sp.	Mexico	1963	Ab
Lump-nosed	*Corynorhinus rafinesquii*	Ft. Detrick, Md.	1956	LE
Mexican fruit bat	*Artibeus jamaicensis*	Mexico	1963	LE
Do	*Artibeus turpis*	----do----	1963	Virus isolation
Short-tailed leaf-nosed	*Carollia perspicillata*	Colombia	1970	Do.
Do	*Carollia subrufa*	Mexico	1966	Do.
Burro, Mexican	*Equus asinus*	West Indies	1962	LE
Cow	*Bovis* sp.	Panama	1959-62	Ab
	----do----	Venezuela	1962-64	Ab
	----do----	Mexico	1963-67	Ab
	----do----	Utah	1965	Ab
	----do----	Texas	1971	Ab
Coyote	*Canis latrans lestes*	Utah	1968	LE
Deer, white-tailed	*Odocoileus virginianus*	Florida	1968	Ab
Dog	*Canis familiaris*	Panama	1959-62	Ab
	----do----	Venezuela	1962-64	Ab
	----do----	----do----	1965	LE
	----do----	Texas	1971	Ab
Donkey	*Equus* sp.	Trinidad	1944	LE
	----do----	Colombia	1958	Virus isolation
	----do----	Venezuela	1962-64	Ab
Fox				
Gray	*Urocyon cinereoargenteus*	Texas	1971	Virus isolation
Kit	*Vulpes macrotis*	Utah	1965	Ab
Goat	*Capra* sp.	Venezuela	1962-64	Ab
	----do----	Texas	1971	Ab
Horse	*Equus caballus*	Venezuela	1939	Virus isolation
	----do----	CDC	1956	LE
	----do----	Guatemala, Honduras	1965-68	Ab
Jackrabbit, black-tailed	*Lepus californicus*	Utah	1965	Ab
	----do----	Texas	1971	Ab
Monkey				
Rhesus	*Macaca mulatta*	Brazil	1960	LE
Tufted capuchin	*Cabus apella*	----do----	1958	LE
	----do----	----do----	1961	Virus isolation
Mouse				
Cotton	*Peromyscus gossypinus*	Mexico	1963-67	Ab
	----do----	Florida	1964	Ab
Deer	*Peromyscus maniculatus*	Utah	1965	Ab
Forest spiny pocket	*Heteromys anomalus*	West Indies	1962	Virus isolation
Laboratory	*Mus musculus*		1960	LE
	----do----		1962	LE
Short-tailed cane	*Zygodontomys brevicauda*	----do----	1962	Virus isolation
Western harvest	*Reithrodontomys megalotis*	Utah	1965	Ab

See footnote at end of table.

Table 13.—Animals associated with Venezuelan Equine Encephalomelitis, 1938-1972[1] *—Continued*

Host	*Species*	*Location*	*Year*	*Test*
Mammals—Continued				
Mule	*Equus* sp.	Trinidad	1944	Virus isolation
Opossum				
Common	*Didelphis marsupialis*	Panama	1959-62	Ab
	-do-	Mexico	1963	Virus isolation
	-do-	Texas	1971	Do.
Four-eyed	*Philander opossum*	Panama	1959-62	Ab
	-do-	Mexico	1963	Virus isolation
	Didelphis sp.	Venezuela	1962-64	Ab
	-do-	Mexico	1963-67	Virus isolation
	-do-	Colombia	1967	Do.
	-do-	Texas	1971	Do.
	-do-	-do-	1971	Ab
Paca	*Agouti paca*	Panama	1959-62	Ab
Pig				
Domestic	*Sus* species	Venezuela	1962-64	Ab
	-do-	Mexico	1963-67	Ab
Guinea	*Cavia cobaya*	New York	1938	LE
Rabbit				
Audubon cottontail	*Sylvilagus audubonii*	Utah	1965	Ab
Desert cottontail	*Sylvilagus* sp.	Texas	1971	Ab
Nuttal cottontail	*Sylvilagus nuttalli*	Utah	1965	Ab
Racoon	*Procyonidae* sp.	Mexico	1963	Ab
	Procyon lotor	Panama	1969	Ab
Rat				
Black	*Rattus rattus*	Panama	1959-62	Ab
	-do-	Mexico	1963-67	Ab
	-do-	Florida	1964	Ab
Chisel-toothed kangaroo	*Dipodomys microps*	Utah	1965	Ab
Cotton	*Sigmodon hispidus*	Panama	1959-62	Virus isolation
	-do-	Mexico	1963-67	Ab
	-do-	Florida	1964	Ab
	-do-	Guatemala, Honduras	1965-68	Ab
Laboratory	*Rattus norvegicus*	Brazil	1961	LE
Norway	-do-	Colombia	1967	Virus isolation
Pack	*Neotoma* sp.	Texas	1971	Ab
Rice	*Oryzomys caliquinosus*	Panama	1959-62	Ab
	-do-	Florida	1964	Ab
Roof	*Rattus rattus*	Colombia	1967	Virus isolation
Spiny	*Proechimys semispinosus*	Panama	1959-62	Ab
	-do-	Brazil	1961	Virus isolation
Terrestrial rice	*Oryzomys laticeps*	West Indies	1962	Do.
Sheep	*Ovis* sp.	Venezuela	1962-64	Ab
	-do-	Colombia	1969	Virus isolation
Squirrel				
Antelope ground	*Citellus leucurus*	Utah	1965	Ab
Red	*Sciurus granatensis*	Panama	1959-62	Ab
Variegated	*Sciurus variegatoides*	-do-	1959-62	Ab
Weasel	*Mustela* sp.	Mexico	1963-67	Ab
Birds:				
Ani, groove-billed	*Crotophaga sulcirostris*	Panama	1959-62	Virus isolation
Cardinal	*Richmondena cardinalis*	Louisiana	1955	Ab

See footnote at end of table.

Table 13.—Animals associated with Venezuelan Equine Encephalomyelitis, 1938-1972[1] *—Continued*

Host	Species	Location	Year	Test
Birds—Continued				
Chicken	*Gallus gallus*	Panama	1959-62	Ab
Crow	*Corvus brachyrhynchos*	Louisiana	1955	Ab
Dove, mourning	*Zenaidura macroura*	CDC	1956	LE
Flycatcher, gray-capped	*Myiozetetes granadensis*	Panama	1959-62	Virus isolation
Flycatcher, social	*Myiozetetes similis*	do	1959-62	Do.
	do	do	1966	Do.
Flycatcher, undetermined	*Myiozetetes* sp.	do	1959-62	Do.
Grackle, purple	*Quiscalus quiscula*	Louisiana	1955	Ab
Heron, green	*Butorides virescens*	Panama	1959-62	Virus isolation
Heron, little blue	*Florida caerulea*	do	1959-62	Do.
Herons		Mexico	1963-67	Ab
Heron, yellow-crowned night	*Nyctanassa violacea*	Louisiana	1955	Ab
Ibis, white	*Guara alba*	do	1955	Ab
Jay, blue	*Cyanocitta cristata*	do	1955	Ab
Kingfisher, Eastern belted	*Megaceryle alcyon alcyon*	do	1955	Ab
Robin, clay-colored	*Turdus grayi*	Panama	1959-62	Virus isolation
Sparrow, English	*Passer domesticus domesticus*	CDC	1956	LE
Oriole, black-cowled	*Icterus prosthemelas*	Panama	1959-62	Virus isolation
Sparrow, song	*Melospiza melodia*	CDC	1956	LE
Sparrow, white-throated	*Zonotrichia albicollis*	CDC	1956	LE
Tanager, scarlet-rumped	*Ramphocelus passerinii*	Panama	1959-62	Virus isolation
	do	do	1966	Do.
Tern, Cabot's sandwich	*Thalasseus sandvicensis acuflavidus*	Texas	1971	Do.
Thrasher, sage	*Oreoscoptes montanus*	Utah	1965	Ab
Toucan, keel-billed	*Ramphastos sulfuratus*	Panama	1959-62	Virus isolation
Warbler, myrtle	*Dendroica coronata coronata*	Louisiana	1955	Ab
Wren, cactus	*Camplorhynchus brunneicapillus*	Texas	1971	Ab

[1] The detection in animals of antibodies to VEE is difficult to interpret. Reactions may be due to exposure years in the past or to arboviruses other than VEE. The complement-fixation test does not differentiate between vaccine and epidemic strains of virus. The kinetic hemagglutination-inhibition test has differentiated 8 subtypes in 4 distinct groups capable of evoking different host responses. These antigenic variants, however, are indistinguishable by neutralization test. Consequently, the table presents the detection of antibodies in the various species with no reference to the test used.

For detailed references see *(31)*, *(34)*, and *(17)*.

in rocky crevices, hollow trees, under piles of dead brush, or in burrows. It is active mainly after dark.

The gray fox, *Urocyon cenereoargenteus*, is distributed from southern Canada and eastern, southwestern, and extreme western United States through Mexico and Central America to northern South America. Gray foxes frequent wooded and brushy country and are possibly most common in the arid regions of the southwestern United States and western Mexico.

Urocyon is sometimes called a tree fox because it frequently climbs trees, an unusual habit for the family. The gray fox does not regularly dig a den but occupies cavities and crevices in rocks, caves, and hollow trees. The species is nocturnal, resting during the day among rocks or in thick vegetation *(40)*. The species of Texas wildlife in which VEE virus and antibodies were found during the period June-November 1971 are summarized in table 12.

Bats

Bats, associated with uncleanliness, darkness, and evil, have been objects of revulsion since Biblical times. Perhaps because they were ugly, nocturnal creatures, bats were not the objects of intense scientific interest until the beginning of the 20th century.

In 1908, bats were believed to be vectors of a paralytic disease of cattle, horses, and mules in Brazil. Three years later the disease was diagnosed as rabies, and in 1921 the bat was proven to be a vector. With the extension or recognition of rabies from South America to Central America, and throughout Mexico to the United States, came evidence that bats were implicated both as asymptomatic carriers and long-ranging transmitters of the disease. This further suggested bats as constant reservoir hosts of the virus in nature.

It was noted that a single insectivorous bat consumes its own weight in insects during a 24-hour period. Bats also are hosts to a large number of disease-producing organisms such as trypanosomes, plasmodia, leptospires, treponemes, helminths, and ectoparasites. Bats often associate with mosquitoes in caves, rock piles, and other sites of winter hibernation. Mosquito-bat-mosquito transmission cycles of arbovirus infection have been demonstrated under experimental conditions.

In October 1943, 20 vampire bats *(Desmodus rotundus murinus)* were captured in the VEE epidemic area of Trinidad. It was not known whether any had fed on infected donkeys. Suspensions of the bat brains were injected intracerebrally into guinea pigs, but none developed reactions to indicate the presence of virus. Subsequent studies indicated that the interscapular brown fat may be an important site for the maintenance as well as the proliferation of certain arboviruses. The interscapular brown fat is a vascular, metabolizing tissue that stores glycogen which the bat uses during hibernation *(38)*.

In 1970, 18 bats of three species were collected in an area of epidemic VEE in eastern Oaxaca. Their serum, submaxillary glands, brown fat, brain, and viscera (lung, heart, kidney, liver, and spleen) were examined for virus. Antibodies to VEE as well as a virus resembling type IB were isolated from the viscera of one vampire bat, *Desmodus rotundus, (6)*. This bat had been collected 500 yards from cases of fatal equine encephalomyelitis. Infection could have come from the ingestion of infected blood or from the bite of an infected mosquito.

Vampires inhabit both arid and humid regions from Argentina, Uruguay, and Chile to northern Mexico. They usually reside in cave colonies of several hundred to several thousand bats. Other habitations are hollow trees, old wells, mine shafts, and deserted buildings. In the large colonies vampires have shared retreats with 20 other species of bats.

The usual food of vampire bats is the blood of horses, burros, and cattle, but man and any warmblooded animal may be attacked. Vampires consume 20 to 25 milliliters of blood a day and reportedly cause more than 10,000 equine deaths every year from rabies alone *(6)*.

Insectivorous bats were experimentally inoculated intranasally and intraperitoneally with VEE virus. Despite high concentrations of virus in the blood for 26 days the bats showed no signs of illness. At simulated hibernation temperatures of 10° C. virus titres were low. However, when bats were returned to room temperature after 90 days, titres rose markedly *(7)*.

The absence of ill-effects in the bat, its capability of transmitting disease by bite, and its ability to provide infective blood for vectors of arboviruses satisfy the prime requisites for a reservoir host. Moreover, the hibernation characteristic of some species preserves virus while the migrating species disseminate disease. Vampire bats not only live but cohabit with other species of bats including the Mexican free-tail *(Tadarida brasiliensis mexicana)*, which migrates in large numbers to the southwestern United States every spring *(8)*. The potential for the spread of the disease by bats is enhanced by the knowledge that VEE virus has been isolated from four species, and antibodies (or experimental susceptibility) demonstrated in six additional species of both fruit-eating and insectivorous bats that range from southern Brazil and Peru north to the States of Tamaulipas and Colima, Mexico.

DISCUSSION AND CONCLUSIONS

The virus of Venezuelan Equine Encephalomyelitis, it is theorized, originated in the humid swamplands of the Amazon-Orinoco Rivers. Its detection over the years in areas increasingly distant from its first known foci in tropical South America suggests several possibilities.

First, the dispersal of a prototype VEE virus from the original foci occurred in the remote past. The virus became established in many favorable environments, maintaining itself indefinitely in an arthropod-small mammal cycle. Under certain conditions of climate and population dynamics of arthropods or small-animal hosts, the virus is introduced into an equine. Either as a consequence of continuous multiplication in a variety of wildlife and arthropods, or after its introduction into the equine, an unnatural host, the endemic virus may mutate into a virulent epidemic strain. (Every virus is a genetic derivative of a preexisting virus of similar but not necessarily identical type.) Equines die or become immune and ultimately the epidemic subsides.

The second possibility is that irrespective of the time period in which they were introduced into an environment, endemic viruses remain endemic. Similarly, epidemic strains are always epidemic, their effects on equines depending upon the presence or absence of susceptible animals.

In either case the virus was and is disseminated by many agencies from two distinct ecological environments. The endemic habitat is swamplands and marshes where the season is long, the vegetation lush, and the moisture high. These areas lie along the coasts from South America through Central America and Mexico to the United States. The epidemic strains of virus appear initially in the same marshy environment but then spread into arid, brushy areas. Suddenly the disease appears simultaneously on widely separated premises sometimes in previously affected areas and at other times hundreds of miles apart. This was the pattern that accompanied the recognition of VEE in the United States.

The disease was introduced into Texas by infected arthropods, primarily mosquitoes, from adjacent areas of infection in Mexico extending from the gulf coast northwestward along the Rio Grande River. Aided by favorable southeasterly winds, the range of mosquitoes in migrant flight could have extended considerable distances beyond the border area. Infected horses from Mexico were possible supplemental sources of infection. Insectivorous or fruit-eating bats, as well as migratory birds from the coastal gulf areas of Mexico, may have been involved in the early introduction of VEE virus.

Within the State of Texas the disease was further spread by mosquitoes (and possible biting flies) feeding upon the first viremic horses in and near Brownsville. Commercial vehicles could have assisted in the mechanical transfer of infected mosquitoes to premises over wide areas.

The movement of infected horses before vaccination and quarantines went into effect helped spread the disease from the lower valley to other parts of Texas.

Unburied carcasses lying in the fields for days provided sources for the dispersion of virus by cats, dogs, birds, rodents, and other carrion eaters.

Burning of dead horses may have released aerosols containing virus particles. Southeasterly winds could readily have disseminated virus particles over extensive areas.

Infection may have been spread during the administration of the vaccine.

Some epidemic VEE went unrecognized for lack of clinical signs or laboratory test. The preponderance of morbidity and mortality ascribed to VEE, however, was the sequela of malnutrition, other infections, and conditions of subhealth.

"Disease", said Charcot, "is very old and nothing about it has changed. It is we who change as we learn to recognize what was formerly imperceptible." Mosquitoes infected with epidemic VEE virus were present in the United States in late June 1971. Events in the lower Rio Grande Valley of Texas during June and early July 1971 support the belief that horses were also infected at some indeterminate time before VEE was recognized. Though the extent of residual VEE in nature has not yet been established, it is likely that the infection is being harbored by some native wildlife host or hosts.

Aerial chemical sprays will control arthropod vectors and repeated immunizations will protect equines. Reduction of the threat of VEE to its lowest level, however, requires recognition of the interrelationship of the agencies and associations of the disease in ecological systems. More than that, the existence of an extensive viral reservoir among lower vertebrates; the potential for dissemination by wide-ranging, highly adaptable insect vectors, bats, and migrant birds; the assistance of vehicles, small boats, and airplanes in the mechanical transfer of infected arthropods; and the availability of a large susceptible equine population projects the implications of a realistic control policy beyond international boundaries.

REFERENCES

(1) Beck, C. E., and Wyckoff, R. W. G.
1938. Venezuelan Equine Encephalomyelitis. Science 88:530.

(2) Castillo, E.
1964. Informe sobre una reciente epidemia de encephalitis equina venezolano en la zona norte del estado Zulia. Sanid. Asist. So 29:325.

(3) Chamberlain, R.W., Sikes, R. K., and Nelson, D. B.
1956. Infection of *Mansonia perturbans* and *Psorophora ferox* Mosquitoes with Venezuelan Equine Encephalomyelitis Virus. Proc. Soc. 91:215-216.

(4) Chamberlain, R. W., Kissling, R. E., Stamm, D. D., Nelson, D. B., and Sikes, R. K.
1956. Venezuelan Equine Encephalomyelitis in Wild Birds. Amer. J. Hyg. 63:261-273.

(5) Chamberlain, R. W., Sudia, W. D., Coleman, P. H., and Work, T. H.
1964. Venezuelan Equine Encephalitis virus from South Florida. *Science*, Vol. 145: 3639; pp. 272-274.

(6) Correa-Giron, P., Calisher, C. H., and Baer, G. M.
1971. Epidemic Strain of Venezuelan Equine Encephalomyelitis Virus from a Vampire Bat Captured in Oaxaca, Mexico, 1970. Science 175:546-547.

(7) Corristan, E. C., La Motte, L. C., Jr. and Smith, D. G.
1956. Susceptibility of bats to certain encephalitis viruses. Fed. Proc. 15:584.

(8) Davis, R. D., Herreid, C. F., II, and Short, H. C.
1962. Ecol. Monogr. 32:311.

(9) Davis, M. H., Hogge, A. L., Jr., Corristan, E. C., and Ferrell, J. F.
1966. Mosquito Transmission of Venezuelan Equine Encephalomyelitis Virus from Experimentally Infected Dogs. Amer. Jr. Trop. Med. Hyg. 15:227.

(10) de Mucha Maciás J.
1966. Encefalitis Equina de Venezuela en Tamaulipas, Mexico. Rev. Invest. Salud. Publ. (Mex) 26:277-279

(11) Eddy, G. A., and Johnson, K. M.
1971. An Overall View of Venezuelan Equine Encephalomyelitis in the Americas. (a report presented at an international roundtable on VEE, Mexico, May 12-14.)

(12) Ehrenkranz, N. J., Sinclair, M. E., Buff, E., and Lyman D.
1970. The Natural Occurrence of Venezuelan Equine Encephalitis Infection in the United States—First Case and Epidemiological Investigations. New Eng. J. Med.:282:6:298-302.

(13) Ehrlich, R., and Miller, S.
1971. Effect of Relative Humidity and Temperature on Airborne Venezuelan Equine Encephalitis Virus. App. Microb. 22(2):194-199.

(14) Franck, P. T., and Johnson, K. M.
1971. An Outbreak of Venezuelan Equine Encephalomyelitis in Central America—Evidence for Exogenous Source of a Virulent Virus Subtype. Amer. J. Epid. 94:487-496.

(15) Galindo, P., and Grayson, M. A.
1971. *Culex (Melanoconion) aikenii:* Natural Vector in Panama of Endemic Venezuelan Encephalitis. Science 172:594-595.

(16) Gilyard, R. T.
1945. A clinical Study of Venezuelan Virus Equine Encephalomyelitis in Trinidad, B.W.I. J. Amer. Vet. Med. Assoc. 818:267-277.

(17) Grayson, M. A., and Galindo, P.
1968. Epidemiologic Studies of Venezuelan Equine Encephalitis Virus in Almirante, Panama. Amer. J. Epid. 88:80-96.

(18) Henderson, B. E., Chappell, W. A., Johnston, J. G., Jr., Sudia, W. A.
1971. Experimental Infection of Horses with Three Strains of Venezuelan Equine Encephalomyelitis Virus. Amer. J. Epid. 93:194-211.

(19) Johnson, C. G.
1969. Migration and Dispersal of Insects by Flight. Methuen and Co. Ltd., London, 763 pp.

(20) Jones, R. H., Potter, H. W., and Rhodes, H. A.
1971. Investigations at VEE Epizooties. In 4th Quarterly Report of 1971, Insects Affecting Man and Animals Branch, Livestock Insects Investigations, Kerrville, Tex.

(21) King, W. V., Bradley, G. H., Smith, C. N., and McDuffie, W. C.
1960. A Handbook of the Mosquitoes of the Southeastern United States. Agric. Handbook No. 173, Agric. Res. Service, USDA.

(22) Kissling, R. E., Chamberlain, R. W., Nelson, D. B., and Stamm, D.D.
1956. Venezuelan Equine Encephalomyelitis in Horses. Amer. J. Hyg. 63:274-287.

(23) Kubes, V., and Rios, F. A.
1939. The Causative Agent of Infectious Equine Encephalomyelitis in Venezuela. Science 90:20-21.

(24) Landinez, E. P.
1970. (National Health Laboratory, Bogota, Colombia) personal communication Feb. 3.

(25) Lord, R. D.
1970. Some Vertebrate Host Aspects of Arbovirus Ecology. J. Wild Dis. 6:236-238.

(26) Mackenzie, R. B.
1971. Venezuelan Equine Encephalitis in Colombia, 1967-1971, Observations Regarding Natural History and Vaccines. (A report presented at an international roundtable on VEE, Mexico, May 12-14)

(27) McKercher, D. G.
1959-60. Newer Concepts of Viruses and Viral Diseases. Iowa State Vet. XXII: 21-27.

(28) Murphy, R. C.
1936. Oceanic Birds of South America. Amer. Mus. Nat. Hist., New York, 1:1142-43.

(29) Reta, G. A.
1971. The Problem of Venezuelan Equine Encephalitis in Mexico. (A report presented at an international roundtable on VEE, Mexico, May 12-14).

(30) SAG
1970. Information on the Outbreak of the Epizootic of Venezuelan Equine Encephalitis, July-August 1970 (General Bureau of Animal Health, Ministry of Agriculture and Livestock, Mexico).

(31) Scherer, W. F., Dickerman, R. W., La Fiandra, R. P., Wong-Chia, C., and Terrian J.
1966. Ecologic Studies of Venezuelan Encephalitis Viruses in Southeastern Mexico, Part IV, Infection of Wild Mammals. Cornell Univ. Med. College, Dept. of Microbiology.

(32) Scherer, W. F., Dickerman, R. W., and Ordenez, J. V.
1970. Discovery and Geographic Distribution of Venezuelan Encephalitic Virus in Guatemala, Honduras, and British Honduras, During 1968-69, and Its Possible Movement to Central America and Mexico. Amer. J. Trop. Med. Hyg. 19:703-711.

(33) Sellers, R. F., Bergold, G. H., Suarez, O. M., and Morales, A.
1965. Investigations During Venezuelan Equine Encephalitis Outbreaks in Venezuela 1962-1964. Amer. J. Trop. Med. Hyg. 14:460-469.

(34) Sidwell, R. W., Gebhardt, O. P., and Thorpe, B. D.
1967. Epidemiological Aspects of Venezuelan Equine Encephalomyelitis Virus Infections. Bact. Rev. 31:65-81.

(35) Sotomayor, C. G.,
1946. A Study of the Virus of Equine Encephalomyelitis in Ecuador. J. Am. Vet. Med. Assoc. 109:478-480.

(36) Spertzel, R. O., and McKinney R. W.
1970. VEE, A Disease on the Move. (Presentation at 74th annual meeting United States Animal Health Assoc., Phila, Pa. Oct. 1970.)

(37) Sudia, W. D., Newhouse, V. F., and Henderson, B. E.
1971. Experimental Infection of Horses with Three Strains of Venezuelan Equine Encephalomyelitis Virus. Part II. Experimental Vector Studies. Amer. J. Epid. 93.

(38) Sulkin, S. E.
1962. The Bat as a Reservoir of Viruses in Nature. Prog. Med. Virol. Karger, Basel/New York, 4:157-207.

(39) Vector Control Briefs,
May 1969. Venezuelan Equine Encephalitis Outbreaks in Colombia. South America-1967. HEW, PHS: 14.

(40) Walker, G. P.
1964. Mammals of the World. The Johns Hopkins Press, Balt.: 1148.

(41) Work, T. H.
1964. Serological Evidence of Arbovirus Infection in the Seminole Indians of Southern Florida. Science 145:270-272.

(42) Young, N. A., and Johnson, K. M.
1969. Antigenic Variants of Venezuelan Equine Encephalitis Virus: Their Geographic Distribution and Epidemiologic Significance. Amer. J. Epidem. 89:286-307.

(43) Zarate, M. L., and Scherer, W. F.
1968. Contact-Spread of Venezuelan Equine Encephalomyelitis Virus Among Cotton Rats Via Urine or Feces and The Naso-or Oropharynx. Amer. J. Trop. Med. 17:894-899.

APPENDIX

Historical Chronology

1935	An encephalitic disease of equines is reported in four departments of Colombia.
1936	The disease appears in northern Colombia and later on La Guajira peninsula. Subsequently, the disease is noted on the Venezuelan side of La Guajira, northwest of Lake Maracaibo.
1937-39	The outbreaks spread eastward across Venezuela.
1938	A previously unidentified neurotropic virus is isolated from the brain of a sick horse in Venezuela. The new disease is named Venezuelan Equine Encephalomyelitis.

1941	Encephalomyelitis in equines arises in Ecuador.
1943	Reappearing in western Venezuela, the disease again moves eastward until it is detected in donkeys on the island of Trinidad. The virus of VEE is isolated from a sick donkey.
1946	Equine encephalomyelitis, which has spread along the entire Pacific coast of Ecuador since its probable entry from Colombia in 1941, is identified as identical to the Venezuelan virus.
1955	Antibodies to VEE are detected in migrant waterfowl in Louisiana.
1960	Antibodies to VEE are found among Seminole Indians living north of the Florida Everglades.
1961-62	Encephalitic disease appears in La Guajira and Llanos Orientales, Colombia. The disease again moves eastward across Venezuela. VEE virus is isolated from humans in Venezuela.
1962	Antibodies to VEE are found in a febrile boy living in Champotón on the gulf coast of the State of Campeche, Mexico.
1963	VEE virus is isolated from mosquitoes and antibodies to VEE are detected in cattle and pigs on the gulf coast of Veracruz, Mexico.
1963	A VEE virus is isolated from mosquitoes in the Everglades National Forest, Florida.
1965	Antibodies to VEE virus are detected in wildlife and beef cattle in Utah.
1965	Antibodies to VEE virus are found in humans, horses, and cotton rats in Guatemala and Honduras.
1966	Clinical VEE is recognized in equines in northern Veracruz and southern Tamaulipas, Mexico.
1967	Clinical VEE is reported near Tampico and Tuxpan, Veracruz.
1967-68	VEE appears in the Atlantic Coastal Plains and the lower Cauca and Magdalena River Valleys, Colombia. The Colombian Ministry of Agriculture requests and receives an experimental vaccine (TC-83) developed by the United States Army Medical Research Institute of Infectious Diseases. The vaccine is used on approximately 8,000 equines in 1968.
1969	Epidemic VEE appears on the coastal plain of Ecuador. U.S. Air Force sprays with ultralow volume malathion.
1969	An outbreak of VEE originating between Guatemala and El Salvador spreads along the coast into Honduras and Nicaragua. Epidemic VEE virus, previously unrecorded in Central America, is identified. U.S. Department of Defense furnishes more than half a million doses of TC-83 vaccine, which is credited with helping to halt the epidemic.
1969	Clinical VEE occurs in the upper Grijalva River Valley of Mexico near the Guatemalan border.
1970	VEE arises in Costa Rica.
1970	Clinical VEE outbreaks are reported in four municipalities in the State of Chiapas, Mexico. VEE virus is isolated in March 1970. Mexico receives 200,000 doses of TC-83 vaccine. The disease spreads to Oaxaca, Veracruz, Guerrero, and Michoacan.
1970 May	Plans are drafted to evaluate the safety of TC-83 vaccine at the Veterinary Science Research laboratory, Denver, Colo.
1971 January	In response to a request for an opinion from the Director of Science and Education, USDA, the Surgeon General of the Public Health Service states that TC-83 vaccine offers no greater risk than that of epidemic VEE. The Surgeon General recommends stockpiling sufficient TC-83 to use on horses in the United States and continuance of supplying the vaccine to the Latin American countries.

1971 April	Clinical VEE appears in northern Veracruz. Presumptive VEE south of Tampico, Mexico, is diagnosed by an investigator from the Cornell University Medical College. Mexican authorities vaccinate in the area and in a zone extending 120 miles to the north. Epidemic VEE virus is isolated from sick horses and a veterinarian in northern Veracruz.
April	Representatives of Plant Protection, Veterinary Sciences Research, Entomology Research, and Animal Health Divisions, USDA, develop plans to combat VEE.
May 12-14	The entire VEE problem is explored at an international roundtable on VEE in Mexico sponsored by the Mexican Department of Agriculture and the Pan-American Health Organization.
May 14	The Director of Science and Education USDA, requests the Department of Defense to set aside 1 million doses of vaccine for possible use on equines in the United States.
May 23	The United States supplies 200,000 doses of vaccine for use by Mexico in vaccinating equines from the United States-Mexico border south. The USDA and the General Office of Animal Health of Mexico agree to test safety and efficacy, including back-passage of TC-83 vaccine in horses, at Palo Alto, Mexico.
June 8	The USDA discusses with representatives of the equine industry alternative plans to control VEE.
June 12	The vaccination program begins in northern Tamaulipas, Mexico.
June 18	The shipment of horses from Mexico to the United States is suspended.
June 19	A multi-disciplinary task force to combat VEE assembles at Harlingen, Tex. The force includes representatives of the U.S. Department of Agriculture, the U.S. Public Health Service, the Texas Animal Health Commission, and the Texas State Department of Health.
June 23	Following agreement between the State of Texas and the USDA, TC-83 vaccine is released for controlled use by veterinary practitioners in 13 counties in Texas south of a line from Corpus Christi to Laredo.
June 25	The voluntary vaccination program begins.
June 25	Sick horses are reported in Ejido Palo Blanco, 7 miles south of the Rio Grande River. Epidemic VEE is subsequently confirmed at CDC, Atlanta.
July 3	The first 50,000 doses of vaccine manufactured from Department of Defense protocols by the National Institute of Veterinary Research, Mexico, are ready for use in northeastern Tamaulipas.
July 8	The Governor of Texas grants permission for an aerial vector control program in the lower Rio Grande Valley.
July 9	The first case of epidemic VEE in the United States is confirmed by CDC, Atlanta, in a horse near Brownsville, Cameron County, Texas. The State of Texas quarantines the 13-county area.
July 10	The United States Air Force, Tactical Air Command, begins aerial spraying to control mosquito vectors in the Rio Grande Valley. The Plant Protection Division coordinates the vector control program.
July 13	The USDA places a Federal quarantine on the State of Texas. The Texas Animal Health Commission quarantines the movement of all equines within the State.
July 15	Vaccinations begin in Texas outside the original 13-county area.
July 16	The U.S. Secretary of Agriculture declares a national emergency from the threat of VEE and provides funds for expanded vaccination and vector control programs. An additional 1.8 million doses of vaccine are requested from Department of Defense.

1971 July 18	Vaccine is made available to the States of New Mexico, Oklahoma, Arkansas, and Louisiana with practitioners on a fee-basis supplying vaccine free.
July 19	A regional VEE control office is opened in Houston to coordinate the new programs. Equines in the states of New Mexico, Oklahoma, Arkansas, and Louisiana are placed under Federal quarantine. Officials of the USDA and of Mexico meet in Matamoros to discuss continued cooperative efforts against VEE.
July 20	The U.S. Air Force completes the first phase of the spray program in Cameron and Hidalgo Counties. The Plant Protection Division coordinates vector control operations at Houston; assembles its field force at three bases of operations; and negotiates contracts with commercial applicators and five county mosquito abatement units.
July 21	The U.S. Air Force begins phase II of the spraying program in Houston area using ultralow volume malathion and dibrom (naled).
July 24	The Veterinary Biologics Division grants a special license for the commercial production of VEE vaccine.
July 25	The Plant Protection Division adds two additional bases of operations at Houston and at Victoria, Tex. Fee basis vaccination is extended to California, Arizona, Mississippi, Alabama, Georgia, and Florida.
August 1	Officials of the USDA and of Mexico meet in Brownsville to discuss progress of the VEE program.
August 3	Mississippi is placed under Federal quarantine.
August 7	Texas requires vaccination of all horses. The Air Force completes its spray mission in the Houston area. All planes and crews are released.
August 8	Private contractors begin the second spray application at Houston.
August 13	The "experimental" classification of TC-83 vaccine and the use of waivers of claim are discontinued.
August 14	Mosquito abatement operations are completed. All commercial aircraft are released. Support to mosquito abatement districts is terminated.
August 20	The Regional VEE Control office at Houston is closed. Operations are transferred to the Ready Room, Beltsville, Md.
August 24	Vaccination on a Federal fee basis is extended to Kentucky, Tennessee, North Carolina, South Carolina, Virginia, Maryland, Delaware, New Jersey, and the District of Columbia (total 19).
September 10	Federal quarantines are removed from Arkansas, New Mexico, and Oklahoma.
November 9	Federal quarantines are removed from Mississippi.
November 11	Epidemic VEE is confirmed in Starr County, Tex. making this the 26th county and the 75th case of epidemic VEE confirmed in the United States.
December 17	The last vaccination program is completed in Texas.
December 31	The Ready Room at Beltsville, Md. is closed.

U.S. GPO 1973/726-657/475/1301